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BRAIN.

PART 2, VOL. 59.

THE MOTOR CORTEX IN MAN IN THE LIGHT OF HUGHLINGS JACKSON'S DOCTRINES.

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The ninth Hughlings Jackson Lecture delivered before the Section of Neurology of the Royal Society of Medicine, on July 31, 1935.

HUGHLINGS JACKSON was the first to point out that there is such a thing as a motor cortex. He was the first to state clearly that the brain, the organ of mind, possesses motor functions. He discovered and elucidated this fact long before further evidence of the motor functions of the cerebral cortex was provided by the experimental investigations of Hitzig and Ferrier on animals. Jackson says: "The convolutions of the brain must contain nervous arrangements representing movements. There is nothing else they can represent except movements and impressions."

He was lead to this conclusion partially by the more general reflection that even the highest nervous centres are only developments of lower centres, which no one doubts represent movements and impressions, a conception in which, as he says, he followed Laycock and Herbert Spencer's doctrine of evolution. But the full evidence of the motor functions of the cerebral cortex was laid bare to him by what he called the experiment made on the brain by disease. That each disease is to be considered as an experiment in the case of man, that it is to be studied anatomically and physiologically, is one of the most important Jacksonian principles, a principle which, at least in Neurology, was not stated nor clearly expressed before his day. He says: "Wherever there is nervous tissue, disease may occur. Now disease can lead to either of two kinds of changes in this tissue, either to destruction of function or to instability, to an over-readiness to discharge. There are destroying lesions and discharging lesions, there is loss of function or over-function. Epileptic convulsions are movements. Although the development of movements occurring during an

attack of convulsions be very abrupt and the number of movements developed at once be so great that the visible result is apparently a mere heedless struggle of muscles, epileptic convulsions are movements—caricatured movements. Epileptic convulsions are nothing but occasional abrupt and excessive discharges of motor nerve-cells, of cells the function of which is mediating efferent impulses to muscles. Now in many cases of epileptic convulsions there is a lesion in the cerebral cortex, for instance a tumour, or a syphiloma, or a tuberculoma or a vascular lesion. Whatever may be the pathological nature of such a lesion, the essential cause of the convulsions is the unstable equilibrium of the nerve cells of the cortex. Now as the cortical grey matter responds to those irritating lesions by convulsions, that is to say by movements, the convolutions of the brain must contain nervous arrangements representing movements, the convolutions must possess motor functions."

Nowadays this sounds like a truism. However, before Hughlings Jackson no one had realized it. Jackson's ideas were revolutionary. In his days the prevalent view as to the cause of epilepsy was that it depended upon disturbances of the medulla or the pons, and nobody believed that the cortex of the brain, the organ of mind, contained motor nervous elements and that an epileptic convulsion was nothing but a sudden, rapid and excessive discharge of these cortical motor cells.

Jackson's view was that the whole cortex of the brain contains nervous arrangements representing movements and impressions. But different parts of the brain represent movements and impressions in different ways and in different combinations. He differentiated the *motor cortex proper*, that is to say those cortical areas which are by the way of efferent pathways in direct or indirect connection with the motor nuclei of the spinal cord, from the *superimposed cortical areas*, which, although they contain similar nervous arrangements re-representing movements and impressions, are in connection with the peripheral endorgans *only* by the intermediary of the motor cortex proper. Adapting his terminology to the principle of evolution and to his conception of the hierarchy of the nervous system, Jackson called the motor cortex proper the *middle level* and the superimposed cortical areas *the highest levels*.

I shall not deal in this lecture with the anatomical aspects of the motor cortex, with the specific cyto- and myeloarchitectonic structures of the different areas of which it is composed, and the anatomy of the cortico-spinal pathways. There is one point only to which I must call attention, that is, that Hughlings Jackson had already recognized

the existence of the two systems connecting the motor cortex with the spinal anterior horn cells, the direct cortico-spinal pyramidal tract and the many articulate cortico-subcortico-spinal pathways.

Fig. 1 demonstrates the extent of the motor cortex proper of the human brain. It shows the different cortical areas from which motor responses are to be obtained by electrical stimulation. The diagram is the outcome of electrical explorations of almost 300 human brains exposed by operation. In the majority of the cases the exploration was made under local anæsthesia.

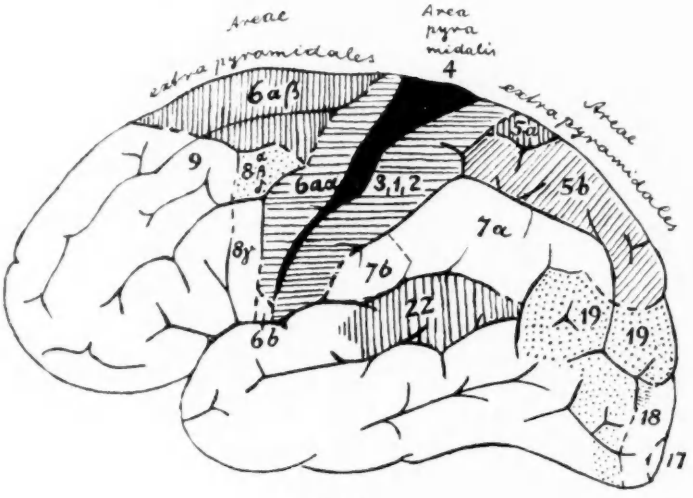

FIG. 1.

In the centre of the motor cortex lies the *anterior central convolution*. It is characterized by two physiological ear-marks, first a low threshold of excitability and secondly its highly differentiated focal subdivisions. In each focus a different part of the body is represented. The stimulation of a given focus produces a single isolated movement of the corresponding part of the body.

The arrangement of the foci in the anterior central convolution from the superior border of the hemisphere down to the Sylvian fissure is well known. At the superior border of the hemisphere the precentral convolution extends to the mesial surface into the paracentral lobule, which contains the foci of the foot, of the toes, of the bladder, and of the rectum.

Nowadays the knowledge of the focal subdivisions of the precentral convolution is common to all neurologists, but Jackson had the same conception as we have to-day. Had he taken a pencil he would have traced the map of the motor cortex with the same accuracy and detailed exactness as it can be done to-day upon the basis of many hundreds of electrical explorations. Jackson did not use the electrical experimental method, he analysed with his keen eyes and his ingenious intuition experiments made by disease on the human brain. He says: "When an epileptic convulsion is limited to the big toe, or to the shoulder, or to the thumb (or to the thumb and the index finger), the seat of the discharging lesion must be different in each of these attacks. In the first case in which the convulsion is limited to the big toe, the discharging lesion must be in that part of the brain in which the big toe is represented preponderatingly. The convulsion limited to the thumb points to the area where the thumb has its main representation.

"When the convulsion begins in the big toe, it spreads secondarily to the foot, next to the calf, then to the upper thigh; the convulsion goes *up* in the leg; thence it spreads to the abdominal wall, to the chest and the shoulder, whence it goes *down* the arm to the fingers, then neck and face follow, first the forehead and the eye-lids, next the lip and the tongue. But when the convulsion begins in the face and spreads to the arm, the fingers are the first to be seized, and the spasm goes *up* in the arm and *down* in the leg, where it terminates in the toes. The part of the body where the convulsion begins indicates the part of the brain where the discharge begins, where the discharging lesion is situated. But from the focus discharging primarily the discharge spreads *laterally* to the adjacent 'healthy' foci. One focus after the other is seized by the radiating waves of impulses. The *march* of the attack, the *order* in which the different parts of the body become involved, reveals the arrangement of the corresponding foci in the precentral convolution."

This principle of localization once being established, one single anatomical observation only was needed. Jackson was provided with it already in 1872. In a case of cortical epilepsy in which all the attacks began in the thumb, and some were limited to it, a tuberculoma of the size of a hazel nut was found just anterior to the precentral convolution in the foot of the third frontal convolution, that is to say just anterior to the focus of the thumb.

All those who read carefully Jackson's descriptions of the march of the convulsions in the case of cortical epilepsy with which he deals,

cannot forbear acknowledging that the picture of the focal somatotopic subdivisions of the anterior central convolution stood clearly before his mental eyes long before the experimental work of Hitzig, Ferrier, Horsley, Sherrington and others on monkeys and anthropoids, and the electrical explorations of the human brain undertaken by neurosurgeons as Horsley, Keen and Mills, Hearn Lloyd and Deaver, Parker and Gotch, Sachs and Gerster, Diller and Buchennan, Nancrède, Harvey Cushing, Fedor Krause, van Valkenburg and others had furnished the stones of the mosaic to which the functional subdivisions of the anterior central convolution can be compared. The map of the human motor cortex to be found nowadays in every textbook or handbook of neurology is nothing else but a copy of the picture engraved in Hughlings Jackson's brain.

I shall quote two cases only from my own material to show the localizing value of the onset of the convulsion and the importance of the march of the convulsion, which reveal in detail the grouping of the foci of the precentral convolution.

Case 1.—A coachman, aged 43, fell from the box of his car and hurt his right temple against a stone. He received no concussion or superficial local wound. Within eight days epileptic convulsions occurred, beginning in the mandible, which was opened and deviated to the left, then the tongue was pulled to the left, the corner of the mouth was drawn to the left and both eyelids were closed, the left more strongly than the right. Thereafter the head turned to the left, then the thumb, fingers, hand, forearm, upper arm and the shoulders, first the left shoulder alone, but within the next second the right one also, were involved in succession. Thereafter the muscles of the chest and the abdominal wall of both sides were seized by spasm, then the left hip became involved and the convulsion spread down the leg to the toes and ended by an involuntary emission of urine and fæces without loss of consciousness. Similar attacks followed, each attack as like the others as one egg to another. Operative exposure revealed a cysticercus embedded in the most inferior portion of the anterior central convolution. It was removed without difficulty, and since then the man has been free of attacks.

In the following case the development of the convulsions was in the reverse order:—

Case 2.—A farmer, aged 43, had suffered from epileptic attacks for eight years. The convulsions began in the big toe, but in some of the attacks involuntary urination was the first symptom. From the toes the convulsion spread to the foot, calf, upper thigh, abdominal wall, chest, and the shoulder, and down the arm to the fingers and the thumb. Then the neck, face, tongue, mandible and palate were involved, and the attack terminated by a severe

spasm of both vocal cords which almost suffocated the man. After each attack there was a postparoxysmal paralysis of the leg.

In this case I found at operation a small soft tumour in the cingulate gyrus involving to some extent the paracentral lobule. The tumour was removed, and on histological examination was found to be a metastatic hypernephroma. It is noteworthy that a metastatic tumour in the brain may produce serious symptoms over a period of eight years, while the primary tumour causes no symptoms at all. Hypernephromas may live in silence and darkness.

Postparoxysmal paralysis, as occurred in this case, was studied carefully by Jackson. He refers to previous observations of Todd and Robertson on epileptic hemiplegia, but Jackson was the first to give the physiological explanation of the paralysis subsequent to epileptic convulsions, an explanation which has been accepted by all succeeding investigators. The paralysis is due to exhaustion of the cortical motor nerve-cells, and on this depends the transient loss of function.

I cannot deal in this lecture with the many interesting physiological details revealed by the electrical exploration of the anterior central convolution in man. I shall content myself to mention those which were foreseen by Jackson.

The first is the relatively large extent of the finger area, in which each single finger has its special representation. Jackson says: "The quantity of cortical grey matter varies not so much with the size of the muscles of a part of the body, as with the number of movements of that part. Thus the small muscles of the fingers will be represented by much more grey matter in the cortex than will be the voluminous muscles of the upper arm, because the former serve in more numerous, different, and in more specialized movements. Greater differentiation of function implies larger representation in the brain."

The second point is the bilateral cortical representation of different parts of the body. Although inspired by the hypothesis of Broadbent, Hughlings Jackson was the first to realize that not only those muscles which, as a rule, act bilaterally, as the respiratory muscles, the muscles of the abdominal wall, and the muscles of the forehead and the eyelids, have a bihemispherical representation, that is to say, those of the two sides when acting together are represented in each of the two hemispheres, but also that every other muscle group is represented not only in the contralateral hemisphere, but to a certain degree in the ipsilateral hemisphere also. Jackson was led to this conclusion by analysing the march of the convulsions in epileptic attacks which begin unilaterally, but involve at the end of the seizure all parts of the other half of the body as well.

The results of the electrical stimulation of the precentral gyrus in man corroborate strongly Jackson's view. Some foci, for instance those of the larynx and palate, respond even to stimuli of lowest intensity by bilateral effects only; both vocal cords are adducted and the palate is elevated without lateral deviation. When from the focus of the mandible closing of the jaw is obtained, the contractions of the masseters and temporal muscles are equally strong on both sides, but when the mandible is opened it deviates to the contralateral side in consequence of the isolated action of the ipsilateral external pterygoid. The foci of the forehead, of the eyelids, of the shoulder, of the chest and of the abdominal wall respond only unilaterally to threshold stimuli, but the slightest increase of intensity of the stimulus produces bilateral effects. By using stronger stimuli bilateral effects can be obtained from almost any focus, but more easily from the foci of the proximal segments of the extremities than from those of the distal segments. I have observed ipsilateral movements of the fingers in a few cases and only when using very strong faradic currents. According to Jackson the movements of the fingers are the most specialized, the most differentiated, the least automatic movements; they have the least bilateral cortical representation.

It is noteworthy that bilateral effects can still be obtained from the precentral convolution after the corpus callosum has been transected. In several cases of obstructive hydrocephalus in which I divided the corpus callosum for a length of 6 to 7 cm., faradic stimulation of the leg area produced similar bilateral movements of the legs as before operation. These observations confirm admirably Jackson's view that the two halves of the body are represented in each of the two hemispheres.

The third point of interest is the *overlapping* of the foci. It has been assumed that the precentral gyrus is composed of distinct and separate foci, each of which corresponds to a special part of the body *exclusively*, and that each focus in which a single segment of the body is represented contains distinct and separate sub-foci for the different movements of that segment, as flexion, extension, abduction and adduction, but such a formula of extreme localization would never have been accepted by Jackson. He was, as he says, neither a fanatic localizer nor was he a universalizer. According to his doctrines a single part of the body, let us say the thumb, is represented *preponderatingly* in one part of the cortex, but it is represented in other parts of the precentral convolution as well, although in a different degree and in different combinations with other parts of the body. All who have performed many electrical

stimulations of the human brain must have become aware of the inconstancy of the effects produced by repeated stimulation of one and the same spot of the precentral gyrus. Let us choose an example. A given spot of the thumb-focus is stimulated repeatedly at intervals of one second by galvanic threshold stimuli. The first, second, third and fourth stimulations result in a movement of the thumb. On the fifth stimulation this movement is fading. The sixth stimulus produces not a movement of the thumb, but a movement of the index finger. The same result is obtained by the seventh stimulus. On the eighth stimulation all fingers except the thumb move. On the ninth stimulation the finger movements are hardly visible. On the tenth and eleventh stimulations the hand moves, and on the twelfth stimulation the primary effect, the movement of the thumb, reappears and is obtained by subsequent stimulations. All these different effects are obtained from one and the same spot and with equal current-intensity. Thus the so-called thumb-focus is not an absolute focus; it contains elements representing the thumb, the fingers and the hand, intermixed with each other. The elements of the thumb are numerically far superior to the other elements and they have the lowest threshold; therefore they are the first to respond to stimulation of the focus. But if the electrical stimulation, which is an un-physiological stimulus, be repeated, the elements of the thumb, after having discharged five to six times, become exhausted and the finger elements lying in the same spot begin to respond. As their threshold is higher than the threshold of the thumb elements, they did not respond to the first stimulus, these are in fact inhibited by simultaneous induction brought about by discharge of the thumb elements. But after the latter have become exhausted the finger elements are in the condition of post-inhibitory exaltation and consequently respond to stimuli previously ineffective. When the finger elements have become exhausted the hand elements, lying in the spot, respond. Finally they become exhausted in their turn. But in the meanwhile the thumb elements have stored up new energy, they have recovered from their exhaustion and have regained their previous reactability.

The inconstancy of the effects and the variation of the responses of one and the same spot to repeated stimulations can be demonstrated in almost every focus of the precentral convolution. Each focus contains not only elements of the part of the body represented preponderatingly in that focus, but also elements of other parts of the body. The foci of the different parts of the body are not like the stones of the mosaic

to which they were compared, but they overlap to a more or less considerable degree. The anterior central convolution does not resemble a painting of cubistic style, it reveals rather the intimate mixture of soft colours and smooth forms of a Raphaelitic Madonna.

According to the Jacksonian method of studying the functions of nervous substrata we have not only to regard the phenomena brought about by stimulation of the substratum and the symptoms resulting from its discharge, but we must also take account of the sequels of the destruction of the substratum. In Jacksonian terms we have to analyse the symptoms of discharging lesions and of destroying lesions. The physiological stimulation of the exposed human brain and the physiological experiment made on the human brain by discharging lesions have given us a clear insight into the organization of the precentral convolution; they have revealed its highly differentiated focal somatotopic subdivisions. Do the results of destroying lesions corroborate the conclusions deduced from the results of stimulation and discharge?

Since the classical investigations of Charcot, Nothnagel and many others, cortical crural monoplegia, brachial monoplegia, and monoplegia of face and tongue as the results of destruction of the three main areas of the precentral convolutions are common knowledge. Destroying lesions limited to small parts of the three main areas give rise to paralysis limited to a single segment of the extremities or to a single part of the body. Let me quote a few examples of such local paralysis.

(1) A bilateral traumatic lesion of the paracentral lobules, due to a fragment of shell-casing, produced paralysis limited to both feet and the toes. The movements of the leg and the thigh were not interfered with. There was also loss of voluntary control over the bladder and rectum. Bilateral lesions of the paracentral lobules often give rise to vesical and rectal disturbances.

(2) A tuberculoma of the right paracentral lobule caused paralysis of the left foot and the toes. The movements of leg and thigh were unimpaired.

(3) In a case of cortical epilepsy I excised the leg area and the trunk area. The incision passed just through the focus of the shoulder, and resulted in paralysis of the right leg, of the muscles of the abdominal wall, of the spine and of the chest on the right side, of the right side of the diaphragm and of the right shoulder. The arm, hand and fingers remained free of motor disturbances.

(4) A shell-fragment injured the anterior central convolution 4 cm. below the superior border of the hemisphere. There was paralysis of the right shoulder, the right upper-arm and forearm. The movements of the hand and the fingers were intact, even supination of the hand and opposition of the thumb and all the delicate movements of the fingers, which suffer first and most severely when the hand-finger area is damaged.

(5) In the literature a good many cases of isolated paralysis of the hand and fingers, of the fingers alone, of one or two fingers, or of the thumb alone, are described as the results of small lesions within the cortical finger area. I have seen many similar cases.

(6) In a patient who suffered from Jacksonian epileptic convulsions three small tuberculomas were excised, together with the surrounding brain tissue, from the precentral convolution. The excision was followed by paralysis of the interossei and lumbricales with the typical claw position of the fingers. All the other muscles of the fingers and thumb acted well. I have observed several other cases of similar cortical paralysis of the interossei.

(7) As a result of a traumatic lesion of the most inferior portion of the precentral gyrus, the patient suffered from epileptic convulsions beginning in the tongue and the face. The foci of the larynx, the palate and the mandible were destroyed, and owing to paralysis of the left external pterygoid, the mandible when opened deviated to the left. The movements of the larynx and palate were not affected since they have bilateral representation.

These examples show that small destroying lesions within the precentral gyrus result in paralysis limited to distinct parts of the body. They show just as well as the electrical stimulation and Jacksonian cortical convulsions the differentiated somatotopic subdivisions of the precentral convolution. The character of the cortical paralysis will be analysed later. At the moment I wish only to call attention to the fact that such a focal paralysis is accessible to repair to a considerable degree. The restitution depends on compensation which is furnished, first, by the cortical elements lying in the adjacent unimpaired foci, and secondly by the ipsilateral precentral convolution. The smaller the area destroyed, the more complete the restitution.

According to the anatomical investigations of Campbell, Brodmann, Vogt and Economo, the precentral gyrus of the human brain is composed of two areas of different cyto- and myeloarchitectonic structure, of Area 4 or the area giganto-pyramidalis, and Area 6 aα or the area agranularis centralis. The extent of Area 4, which is marked black in fig. 1, is characterized by the presence of the Betz cells in the fifth cell layer. The size of the Betz cells diminishes gradually in the descending direction. This fact, well known to-day, was foreboded by Jackson. In a paper published in 1874 he says: "I should, for theoretical reasons, expect that of the centres those which represent movements of the small muscles (eyes, face, fingers, hand) will have cells comparatively small, smaller than the cells superintending movements of the large, more voluminous muscles of the limbs and the trunk." A few years later, in 1879, Jackson's opinion was confirmed by the anatomical researches of Bevan Lewis.

We come now to the question as to whether there are physiological differences between the two areas composing the precentral gyrus in man, Area 4 and Area 6 a*a*. The opinion has been recently expressed that the different cyto- and myeloarchitectonic structure of these areas were merely anatomical details without physiological significance. Hughlings Jackson was of other opinion. He says:—

"Differentiation of function implies differentiation of structure and differentiation of structure means differentiation of function. So far as the comparative anatomist detects homological cerebral structures, so far, it is to be hoped, will the physiologist be able to develop the homological *functions*, and so far as the anatomist ascertains *different structures*, so far will the physiologist be able to detect different functions. Since, as Lockhart Clarke has pointed out, the structure of the anterior convolutions does differ from the posterior, they must serve differently in mind."

Physiologists have attempted to analyse the functional differences of Area 4 and Area 6. I refer to the fundamental work of the Vogts and of John Fulton and his school. In man Area 4 and Area 6a*a* differ from each other by their threshold of excitability. Both areas respond to electrical stimulation by the isolated movements of a part of the body or of a single segment of the extremities. But to obtain these effects from Area 6a*a* a considerably stronger stimulus is needed than in Area 4. The difference of the threshold amounts to 1 to 2 ma. Under general narcosis Area 6a*a* becomes inexcitable to the galvanic current, and under deep narcosis even to faradic currents. Area 4 preserves its excitability under these circumstances.

As Vogt has demonstrated, the isolated movements obtained from Area 6 a*a* do not depend on motor impulses mediated by the direct efferent tract of Area 6, but on impulses transmitted from Area 6 to Area 4 by the superficial cortical cell layers. From Area 4 the impulses are conveyed to the muscles by the pyramidal tract. After excision of Area 4, or after transection of the superficial cortical layers of Area 6, isolated movements are no longer obtained by electrical stimulation of Area 6. This can be demonstrated in man. When Area 4 is excised, or when the pyramidal tract is interrupted, electrical stimulation of Area 6a*a* fails to produce isolated movements. When the superficial cortical layers of Area 6a*a* are cauterized by electro-coagulation, isolated movements are no longer obtainable from Area 6a*a*, but they can be produced promptly by stimulation of Area 4.

If Area 4 be excised, or the pyramidal tract be interrupted, Area 6a*a*

still responds to strong faradic stimuli, not however by isolated movements, but by mass movements of the whole contralateral half of the body, which I shall describe later when dealing with Area 6aβ. These complex movements depend on impulses originating in Area 6aα and mediated by its own efferent pathway to the subcortical centres, and through them to the spinal motor nuclei.

We come now to the *postcentral convolution*. It is the main cortical area of sensibility. Of this fact Jackson was well aware, though he never believed that any area of the cortex was purely motor or purely sensory. Each cortical area represents movements and impressions, one area preponderately movements, another preponderately impressions. The precentral areas are chiefly motor, the postcentral convolution is chiefly sensory. The exactness of this Jacksonian statement can be demonstrated beautifully by electrical stimulation of the human retrocentral convolution under local anæsthesia. Galvanic currents produce the same isolated movements as are obtained from the precentral convolution, but the threshold is considerably higher; the difference amounts to 2 to 4 ma. Under general narcosis the galvanic currents fail to produce motor effects, and if the narcosis be deep even strong faradic currents are ineffective.

The isolated movements obtained from the postcentral convolution are brought about by the intermediary of Area 4. The impulses are transmitted from the postcentral gyrus to Area 4 by the U-fibres connecting both central convolutions. If Area 4 be excised or the pyramidal tract be interrupted, the retrocentral gyrus no longer responds with isolated movements. Under these circumstances strong faradic stimuli produce mass movements of the whole contralateral half of the body similar to those obtained from Area 6aα when the pyramidal tract is interrupted. They will be described later.

As has been said, the postcentral convolution is the main cortical sensory area. It is composed of highly differentiated foci in which the different parts of the body have their sensory representation. In the posterior part of the paracentral lobule, the structure of which is homologous to that of the postcentral gyrus, the bladder and rectum are represented. Then follow the sensory foci of the different parts of the body from the toes up to the head. Faradic stimulation of any focus is attended by a sensation in the corresponding part of the body. (Galvanic stimuli are ineffective.)

The epileptic attacks resulting from discharge of the postcentral convolution are characterized by sensory auræ. The "subjective

sensation," to speak in Jacksonian terms, begins in that part of the body which is represented in the focus which discharges primarily. From the part where it begins the sensation spreads like a wave successively to the other parts of the body in the order corresponding to the grouping of the foci in the postcentral convolution. The discharge may be limited to the sensory elements so that the epileptic attack consists in subjective sensations only. But as a rule the sensory aura is followed by motor discharges which show the same march as do the convulsions originating from the precentral gyrus. All these details were well known to Hughlings Jackson. He describes beautifully the warning, the crude sensations, the feeling of numbness, of tingling, or of pain. He explains them as being mental states attending the discharge of the cortical sensory elements. With a simply astonishing exactness he described in 1863 the order of the spread of the subjective sensations in a case of cortical sensory epilepsy. Another detail often to be observed in postcentral epileptic attacks, was also mentioned by him; the first sign of the motor discharge following the sensory aura may be a long lasting tremor preceding the clonic twitching.

A few of my own observations will illustrate these points.

Case 3.—As a result of a fragment of shell embedded in the posterior part of the paracentral lobule the patient suffered from sensory epileptic fits beginning with a sudden feeling of micturition and tenesmus, followed by a wave of burning and tingling sensations which spread from the toes up to the face and into the buccal cavity. The sensation terminated in the larynx by a convulsive cough. Some of the attacks remained purely sensory, but in the majority the sensory aura was followed by motor convulsions.

Case 4.—A patient with a traumatic lesion of the postcentral convolution involving foci of the fifth and fourth finger suffered from epileptic attacks beginning with a sensory aura in the fingers, which spread over the whole half of the body. The aura was of long duration; the motor discharge which followed was relatively short and often limited to the arm. After each attack there was a complete postparoxysmal hemianæsthesia, a typical example of exhaustion of the postcentral gyrus caused by the preceding long-lasting discharge of the sensory elements.

The last case is interesting from still another aspect. The patient had a traumatic scar in the left fourth finger. By pressure upon the scar the attack could be produced with experimental promptness. I have observed several other cases of the same kind. They belong in the group of reflex-epilepsy, the mechanism of which has been explained in a masterly fashion by Jackson. He describes a patient who cut the palmar surface of his left index finger with a gouge and who nine

months later developed epileptic convulsions always beginning in the left index and middle fingers. Jackson says: "It may be supposed then that in such cases of convulsion there are two factors: (1) a part of the nervous system unstable, and (2) an irritation starting from the finger, which travels to the cortical area being in the condition of unstable equilibrium and then determines the unstable cortical grey matter to discharge. The irritation from the injured finger acts on the cortex only as the explosion of a gun cap does on the charge in the barrel."

According to Jackson the sensory aura of epileptic attacks depends on the discharge of cortical sensory cells. The aura is in itself an epileptic fit. He says: "It is worth mentioning that some patients have a feeling as if a part were convulsed when it does not really move. One of my patients subject to veritable convulsions beginning in the left thumb had sometimes what he called convulsions not to be seen of that part. Another patient had the feeling of convulsion of one side of his face, but looking in a glass he saw that it did not move. No doubt that, if a man, subject to fits beginning in his right thumb, were to lose the right arm by amputation, his fits would still seem to him to begin in his spectral thumb, for some time at least."

Jefferson has recently described such a case which amounts to the experiment referred to by Jackson. A lady who suffered from local sensory fits of painful character, always beginning in the fingers, was advised to have the nerves of the arm injected with alcohol. Had she looked heavenward she would have seen the earnest smile of the father of Neurology, lifting his finger to warn her, but unfortunately the patient did not do so, she accepted the suggestion given to her. What happened exactly is unknown, but the arm became black after the injection and was amputated above the elbow. The sensory fits were entirely and completely unchanged. I have observed a similar case to which I referred briefly some years ago.

We come now to the frontal extrapyramidal Area 6aβ. When this area is stimulated under local anæsthesia by strong faradic currents a complex mass-movement of all parts of the contralateral half of the body is obtained. Head, eyes and trunk are turned to the contralateral side and the contralateral arm and leg execute combined movements. As a rule the arm is raised and abducted, the forearm is flexed, the hand is pronated and the fist is closed or opened. I call this compound movement the flexor synergy of the arm. Less frequently the reverse synergy ensues, the arm is adducted and extended, but the hand is

pronated as it is in flexor synergy. The leg is simultaneously flexed at the hip and knee with dorsal flexion of the foot and toes, constituting a flexor synergy of the leg; or there may be extension of hip and knee and plantar flexion of the foot, an extensor-synergy of the leg.

The motor effects obtained from Area 6aβ depend on the proper efferent tract of this area which mediates the impulses to the subcortical centres and through them to the spinal cord and the muscles. When the precentral convolution is destroyed or the pyramidal tract is interrupted, stimulation of Area 6 aβ produces the same compound effects as are obtained if the precentral convolution be intact.

Epileptic convulsions originating from Area 6aβ have a characteristic aspect. There is no sensory aura: all the muscles of the contralateral half of the body are thrown into spasm almost at once; the head, eyes and trunk are turned to the side opposite to the discharging lesion and the arm and leg are involved in tonic-clonic mass-convulsions; in the arm the flexor synergy generally predominates, while in the leg the extensor synergists prevail. Towards the end of the discharge the flexor and extensor synergies alternate with each other by typical clonic spasms. In some cases the convulsions of the muscles of the spine are so violent that the patient, if standing when the attack starts, rotates two or three times around himself and then falls to the side opposite to the discharging lesion. Several times I have seen a patient, if lying in bed, thrown out of it by the convulsion and rolled over to one side.

The motor effects of stimulation of the *superior parietal lobe, Area* 5, are very similar to those obtained from Area 6aβ. The main difference is that on stimulation of Area 5 the ipsilateral extremities, chiefly the leg, take part in the reaction soon after the onset of the spasms in the contralateral extremities.

The superior parietal lobe is a part of the sensory cortex, but unlike the retrocentral convolution it is not subdivided into separately differentiated sensory foci. The whole contralateral half of the body is represented as an entity, and there is representation of the ipsilateral half as well, although to a smaller degree. Faradic stimulation of the superior parietal lobe evokes a sensation which is localized over the whole contralateral half of the body, and if the stimulation be prolonged the sensation spreads over the ipsilateral half. The paræsthesiæ are often painful. Some of my patients complained of intestinal pain, of an epigastric sensation, or of pain similar to that of angina pectoris.

Stimulation of the superior lip of the interparietal sulcus evokes violent vertigo. The patient sees the objects before him moving

towards the side of stimulation, or he has the sensation of turning towards the contralateral side, although objectively nothing happens and no turning of the head and the trunk really occurs.

In conformity with the sensory-motor functions of Area 5, the epileptic attacks originating from it begin with a sensory aura. Usually a wave of paræsthesia shoots over the contralateral half of the body and radiates soon to the ipsilateral half. In some cases the patient complains of violent gastric pain, and in several of my cases the warning was a sudden attack of vertigo. The motor discharge following the premonitory aura leads to combined flexion of the contralateral arm and leg and rotation of the head and the eyes. A little later the ipsilateral leg is drawn up and then the ipsilateral arm.

The following case is of special interest in this connection :—

Case 5.—A girl, aged 17, when 2 years old had an attack of infantile hemiplegia, which cleared up completely. When 14 years old she began to suffer from epileptic attacks. In each fit the warning was an attack of vertigo; it seemed to her as if objects moved to the left side, after which she had the feeling of being rolled over to the right side. The vertigo was of long duration and was often accompanied by violent nausea. The subsequent motor discharge was of the character described above. I exposed the left parietal lobe and found a large cyst in the interparietal fissure and at the bottom of the cyst a sclerotic scar. Faradic stimulation of the superior lip of the fissure evoked vertigo similar to that of her spontaneous fits. The girl cried aloud: "Hold me, I fall from the table, to the right side." Objectively nothing happened. The same feeling of vertigo was called forth during the excision of the scar. Since the operation, seven years ago, the patient has been entirely free from attacks.

Jackson has repeatedly discussed vertigo as a common warning in epileptic fits. They are among his most luminous dissertations, and I regret that for reason of time I cannot enter upon them in this lecture.

The temporal extrapyramidal area, Area 22, responds to faradic stimulation by the same compound movements as Area 6aβ. The sensory effects evoked by stimulation of the temporal lobe are chiefly acoustic sensations; the patient hears noises, thunder roaring, buzzing, whistling, sometimes voices of obscure character, seldom real words. Penfield describes dizzy feelings and a sensation about the heart on stimulation of the external surface of the temporal lobe. The epileptic attacks originating from the temporal lobe often begin with an acoustic aura.

I shall quote one case only :—

Case 6.—A boy, aged 11, with right-sided hemiplegia resulting from birth

injury to the brain, suffered from epileptic attacks, the warning of which was an acoustic aura. He heard a confused din of voices, sometimes real words, for instance: "Boy come along!" The aura was followed by violent convulsions, in which his head, eyes and trunk were turned to the right, and his right arm and leg were drawn up by tonic-clonic mass convulsions. In some of the attacks the boy rolled over to the right side. The most interesting point in this case was that the attacks were provoked with experimental promptness by a sudden noise, chiefly when it was unexpected; for instance, when the door was opened abruptly or when an object fell upon the floor, or when the boy was called by somebody whom he had not seen. The case is a typical example of acoustic reflex epilepsy. The two factors to which Jackson directed attention are evident in this case: (1) An area of cortex in the condition of highly unstable equilibrium; (2) incoming impulses which act on the unstable cortical area as the explosion of a gun cap does on the charge in the barrel. Faradic stimulation of the first temporal convolution produced an epileptic attack which was of the same character as the spontaneous attacks, including the acoustic aura.

I pass over the cortical area, 6b, the so-called masticatory area, and the oculomotor cortical areas, 8 and 19, the frontal and occipital motor eye-fields.

Destructive Lesions of the Motor Cortex.

If movements are represented in the cortical motor areas it is obvious that destruction of those areas must result in loss of movements, and that destruction of a single area must lead to loss of the special movements represented in that area. As a concrete example the immediate sequelæ of operative excision of the leg area of the precentral convolution are: (1) Total loss of voluntary motility of the contralateral leg; (2) impairment or loss of reflex activity as well. The stretch reflex is abolished, the paralysis is flaccid. The knee-jerk and ankle-jerk were lost in my cases and no plantar response was obtainable. The latter reflex was the first to reappear, within five to ten hours. In every case the plantar response was then extensor. The tendon reflexes returned within two to three days, the stretch reflex proper within eight to ten days. Then the stage of loss of reflex activity merged gradually into the subsequent state of increased reflex activity; the paralysis became spastic.

The increased reflex activity depends on the loss of control exerted by the higher levels upon the lower levels. The cortical motor leg area is provided not only with innervatory elements, which, if active, bring about contraction of muscles, but also with inhibitory elements which control the reflex activity of the spinal cord. If this inhibitory function

of the cortex be lost the spinal cord behaves like a horse which, no longer checked by the bridle, rushes away on a touch of the spur. I fully realize that many arguments have been put forward against this theory, but no one has given a better explanation of reflex over-activity than Hughlings Jackson did by his principle of loss of control.

The immediate result of excision of the cortical leg area is an initial total loss of voluntary motility of the leg. At the first glance this is surprising, as the leg is represented not only in the area excised, but also in many other cortical areas. Why then is the paralysis total? All the different cortical motor areas, the precentral, frontal, postcentral, parietal and temporal, represent a co-operative society, each partner of which contributes a specific, more or less important, quota to the validity of the society (in the case we deal with, to the achievement of voluntary movements). If one partner, especially if such an important partner as the anterior central area, be eliminated abruptly the whole co-operative society becomes functionally impotent in spite of the anatomical integrity of the other partners (in this case the extrapyramidal cortical areas). But this initial inactivity of the co-operative society is transitory, for sooner or later a re-organization of the surviving members—of the cortical motor areas left intact—takes place. They regain their functional capacity and thereby voluntary motility is re-established. But the restitution of motility is not complete. As Jackson has pointed out, the movements of a limb are represented in many different cortical areas, but in each area in a different degree and in different combinations with other limbs. The specific function of Area 4 is the isolated innervation of single muscle groups. The isolated movements of the single segments of the leg are represented in no other cortical area. Therefore the isolated movements of the toes, of the foot, of the leg and of the thigh are abolished by destruction of the leg area in the precentral convolution. The mode of representation of the leg movements in the other cortical areas, the extrapyramidal areas, greatly differs from the representation in the anterior central convolution. The leg movements brought about by the activity of the extrapyramidal areas are compound movements of all segments of the leg, stereotyped synergies, as the flexor synergy and the extensor synergy. As we have seen, these synergies are elicited by electrical stimulation of the extrapyramidal areas, they appear as caricatured mass movements in the convulsions originating from those areas, and we now see that they are the movements which are performed voluntarily by the patient after the leg area in the precentral convolution has been

excised. These synergies are the sole movements which can be performed.

These synergies are unmodifiable, one component being coupled inseparably with the other components. When the patient dorsiflexes his foot, this movement is accompanied by a simultaneous flexion of the knee and the hip. When he flexes his leg, the thigh is flexed and the foot is dorsiflexed. When the leg is raised the knee is flexed and the foot dorsiflexed. Vice-versa voluntary extension of one of the three segments of the leg is associated with extension of the other two.

The restitution of voluntary motility of the leg after its initial total abolition is called by Jackson *compensation*. According to him a nervous substratum which is active in the compensation can only develop functions primarily immanent to it, it can contribute only to the achievement of movements to which it had already contributed under normal conditions; it can contribute to compensation by its specific functions only.

In the case we deal with, that is excision of the motor precentral leg area, compensation depends not only on the extrapyramidal motor areas of the corresponding hemisphere, but also on the leg area of the other hemisphere. It is, I believe, one of Jackson's greatest achievements that he pointed out that more or less all muscle groups of one half of the body are represented in both the contralateral and ipsilateral hemispheres, although to a different degree. Muscles acting chiefly bilaterally, as those of the abdominal wall, of the spine and the chest, of the eyelids and of the mandible, have the most pronounced bi-hemispherical representation. The proximal muscles of the extremities have a greater bilateral representation than the distal muscles.

As soon as the ipsilateral precentral leg area and its efferent tract, the uncrossed pyramidal tract, take part in the compensation, the possibility of innervating single muscle groups is restored, and thereby the stereotyped extrapyramidal synergies, unmodifiable at first, may undergo modification. One component of the synergy can be accentuated relatively to the other components by isolated impulses to the corresponding muscle group. For example, on raising the leg the associated movements of the leg and the foot, although still present, recede into the background. The further the compensatory function of the ipsilateral leg area progresses, the more the ability to perform isolated movements of the single segments of the leg is re-established. But Jackson says: "Compensation is never complete." When the patient who has reached the degree of compensation described attempts

to perform an isolated movement of a single segment of the leg against external resistance, the synergy breaks through immediately. In order to surmount external resistance stronger cortical impulses are needed, and as they cannot be furnished by the ipsilateral leg area alone, the co-operation by the extrapyramidal areas is indispensable.

In bilateral lesions of the leg area the compensation by the ipsilateral leg area fails; the only voluntary movements possible are those of the extrapyramidal synergies. Isolated movements of the single segments of the leg are permanently lost.

The motor disturbances resulting from excision or destruction of the arm-area are analogous to the disturbances of motility of the leg. There is an initial total loss of voluntary movement and of reflex activity of the arm; the paralysis is flaccid. Then the reflexes return and become increased as the paralysis becomes spastic. The restitution of voluntary motility of the arm develops as in the leg. The isolated movements of its segments remain absent, but compound movements represented in the extrapyramidal areas are possible, as the *flexor synergy*, consisting in abduction of the upper arm, flexion of the forearm, pronation of the hand and closing or opening of the fist, and the *extensor synergy* composed of adduction of the upper arm, extension of the forearm, pronation of the hand and closing (or opening) of the fingers. The synergies are unmodifiable, one component is coupled inseparably with the other components. If the arm be raised the forearm is flexed and the hand is pronated at the same time. If the forearm be flexed the upper arm is raised and the hand is pronated. There is a noteworthy similarity between these compound movements performed voluntarily by the patient and the synergies produced by electrical stimulation or by an epileptic discharge of the extrapyramidal cortical areas. Similarly, the components of the extensor synergy are inseparable; if the forearm be extended, the arm is adducted and the hand is pronated; if the arm be adducted, the forearm is extended and the hand is pronated. When later the ipsilateral arm area takes part in compensation and isolated impulses can be forwarded to single muscle groups, the synergies can be modified, one component can be accentuated relatively to the other components; for instance, the arm can be raised to a considerable degree without the forearm being flexed, or the forearm may be flexed without the arm being abducted at the same time.

But even if compensation by the ipsilateral arm area has reached a high degree, the specialized movements of the fingers almost always

remain impaired, because they are represented almost exclusively in the contralateral finger area. The patient can close and open his fist, but he cannot flex a single finger without the other fingers and the thumb being flexed at the same time. On attempting to extend one finger all the others extend. Moreover, he cannot bend a single segment of a finger: for instance, the terminal phalanx of the thumb or the middle phalanx of the index ; when he attempts to do so all the fingers and the thumb flex at all joints. Opposition of the thumb to the other fingers is lost ; when the patient tries to execute this movement the fist closes. Consequently all the finer skilled movements of the fingers, as buttoning, writing, painting and drawing are lost. Every case of excision or destruction of the cortical hand-finger area illustrates the validity of Jackson's principle of Dissolution. The less specialized gross movements, such as closing and opening the fist, are little or not at all impaired ; the skilled, most specialized movements of the fingers, representing the specific functions of the human hand, are profoundly disturbed. They are the first to suffer when a destroying lesion begins to damage the cortical finger area, and they are the last to recover when the pathological process is removed or when compensation develops.

Nevertheless it must be said that in some cases of destruction of the arm-finger area even the finer skilled movements of the fingers may be restored by the compensatory function of the ipsilateral finger area, especially under the influence of systematic treatment by exercises.

The following case is a good example :—

Case 7.—When I first saw the patient the only movements he could perform were the stereotyped extrapyramidal synergies of the arm. Under treatment he soon learned to modify them by the compensatory function of the ipsilateral arm area to such a degree that he could raise the arm without bending the forearm and open and close the fist strongly, but the finer skilled movements of the fingers remained absent. For instance, in order to hold a pencil, he had to grasp it in his fist. After treatment had been continued for three years the patient learned to perform isolated movements of the fingers and thumb ; opposition of the thumb became perfect, he was able to hold a pencil correctly, his handwriting became, although not calligraphic, much better than that of many normal individuals. A post-mortem examination showed complete degeneration of the left pyramidal tract in the medulla.

Case 8.—Another case was that of a boy who suffered from cortical epileptic attacks, resulting from a sclerotic scar, due to infantile encephalitis. The whole cortical hand-finger area was excised and the epileptic convulsions ceased. There was at first a total loss of all voluntary movement of the hand and the fingers, but the boy soon regained gross movements, as closing and opening his

fist, but the more specialized skilled movements of the fingers could not be performed. For instance, when he attempted to oppose the thumb, all the fingers and the thumb flexed. Under treatment by exercises, continued for years, the skilled movements of the fingers also were restored. He learned to oppose his thumb in a perfectly correct way, but this movement was accompanied by a similar movement of his right thumb; the latter constitutes direct evidence of the compensatory activity of the left cortical finger area. All the finer skilled movements of the fingers, as buttoning and writing, were also accompanied by homologous movements of the left hand, and these associated movements never disappeared completely.

Time compels me to pass over the motor disturbances resulting from excision or destruction of the cortical trunk area and the areas of the face, tongue and mandible, although a description of these disturbances would show in a particularly clear light the amazing gift of observation possessed by Hughlings Jackson.

Summing up the motor disturbances resulting from destruction of the anterior central convolution, we can say that the main symptoms are :—

(1) The *negative* symptoms are loss of the isolated innervations of single muscle groups, loss of the most specialized movements, and loss of the faculty of modifying the stereotyped extrapyramidal synergies and of adjusting them to special purposive acts. The loss of function can be compensated to a more or less considerable degree by the ipsilateral precentral gyrus.

(2) The *positive* symptoms are : (*a*) Exhibition of the functions of the intact extrapyramidal cortical motor areas which are no longer under the control of the anterior central convolution and are dissociated from its activity; (*b*) increased spinal reflex activity, the spinal reflex machinery being freed from the control exerted upon it by the anterior central convolution.

We have to consider now the results of destructive lesions of the *extrapyramidal* cortical areas.

Excision of Area 6aβ or 22, which I have performed in about forty cases, either gives rise to no detectable symptoms, or, if there are symptoms at first, they disappear very soon. The initial symptoms which were observed in some of these cases will be described in a later communication, but I may say that in no case were forced grasping or the slightest degree of spasticity observed.

That after excision of Area 6aβ or Area 22, motor disturbances are absent or, if at first present, disappeared within a short time, is surprising.

Both areas certainly possess important motor functions as has been demonstrated by electrical stimulation and by the epileptic convulsions originating from them. Yet destruction of the areas leads either to no detectable disturbances, or to slight and transient disturbances only. The apparent discrepancy is one of the problems discussed and elucidated by Jackson in many of his papers. When the cortical area in which movements are represented discharges, movements are produced, and if the discharge be a sudden violent outburst of excessive impulses convulsions are brought about. But when the same area is put out of function by a destroying lesion, no detectable motor deficiency is to be expected, if the movements represented in the destroyed area be represented in the same, or in approximately the same, combination and order in other cortical areas also. Under such circumstances the loss of function of the destroyed area is compensated by the other areas. Now Area 6aβ, the postcentral area, Area 5 and Area 22 are functionally very much alike. When Area 6aβ is destroyed there are still other extrapyramidal areas available for compensation. The same principle holds good for Area 22. It does not apply to the retrocentral areas, the postcentral gyrus and the superior parietal lobe, which are the main cortical sensory areas; their destruction leads not only to loss of sensibility, but also to motor disturbances due to loss of afferent impulses indispensable for the regulation of efferent cortical impulses. The motor disturbance brought about by lesions of the sensory cortical areas is an ataxia, equivalent to that produced by lesions of the posterior spinal nerve roots or other afferent tracts.

The important part which the extrapyramidal areas play in the performance of movements is best demonstrated by cases with destructive lesions of the precentral convolution in which motility has been greatly improved by the compensatory function of the extrapyramidal areas and in which later, for some reason or another, the functions of these also become impaired.

In one of my cases of traumatic injury of the anterior and posterior central convolutions the initial complete hemiplegia had improved greatly. The patient suffered from epileptic attacks originating from Area 6aβ. After operative excision of this area the patient had a complete hemiplegia, first flaccid, later spastic. Voluntary motility returned, but the high degree of compensation reached before operation was never attained.

Another patient with a large traumatic lesion of the brain involving the central convolutions and the frontal extrapyramidal Area 6aβ,

had a severe right-sided hemiplegia. He could, however, perform the flexor and extensor synergies fairly well. As he suffered from epileptic convulsions originating from the parietal Area 5, I excised that area to its full extent. Although the complete hemiplegia which followed the operation underwent a certain recovery, the motor disturbance remained much more marked than it had previously been. The only extrapyramidal area left intact, Area 22, did not contribute much to the restitution of motility.

Reviewing experiences collected from many hundreds of cases of destruction of the cortical motor areas I can say that the more extensive the lesion, the more severe is the paralysis and the fewer the movements which persist. Jackson has described in a masterly manner the different degrees of hemiplegia and has shown how they can be explained by the the principle of dissolution. The most specialized, most voluntary and least automatic movements are the first to suffer and the last to be restored. The greater the number of areas destroyed in addition to the precentral convolution, the more the movements which are least specialized, least voluntary and most automatic, are involved.

But even if the whole motor cortex of one hemisphere be destroyed there is still the other hemisphere available for compensation. Jackson's view of the bilateral cortical representation of each half of the body, proved to be correct by many observations already referred to, receives further corroboration from cases of congenital or early acquired destructive lesions. Though such lesions may involve the whole motor cortex of one hemisphere, an astonishing degree of motor ability may exist. If the whole motor cortex be destroyed in adults motility is recoverable to a much smaller degree.

The last question to be discussed is the differentiation between Area 4 and Area 6a. The precentral convolution in man is composed of two areas anatomically different, Area 4 and Area 6a*α*. These two areas behave differently when stimulated by the electric current. The question therefore arises as to whether the motor disturbances resulting from isolated destruction of one of these areas differ from those due to destruction of the other. The Area 4-syndrome and the Area 6-syndrome have been carefully analysed in monkeys and anthropoids by John Fulton and his school. In human pathology cases suitable for the analysis of this problem are extremely rare. In one case of excision of the leg area, strictly limited to Area 4, I found the same motor disturbance as those which follow excision of the whole leg area in the precentral convolution, that is to say both Area 4 and Area 6a*α*.

In a second case in which the excision was limited to the foot centre in Area 4, the foot and the toes were completely paralysed at first, the paralysis was flaccid and the Achilles and the plantar reflexes were absent. Four hours after operation an extensor response of the toes could be elicited, and eight hours after operation the Achilles reflex returned. Then ankle clonus and Rossolimo's sign developed, and movements of the foot occurred in association with flexion or extension of the other segments of the limb, but isolated movements of the foot and the toes could not be performed. In walking the foot as well as the leg and thigh were flexed and the toes did not drop or scrape the ground. After treatment by exercise the patient learned to dorsiflex and plantarflex the foot independently of flexion and extension of the other segments of the limb, provided that the muscles did not have to overcome external resistance, but the isolated movements of the toes were lost permanently.

So far as conclusions can be drawn from these two observations, they seem to demonstrate that the motor disturbance resulting from isolated destruction of Area 4 in man does not differ to a detectable degree from that due to destruction of both Areas 4 and 6aα.

I have no cases of excision or destruction strictly limited to the arm and the finger areas. In this region of the precentral convolution Area 4 lies mainly or exclusively in the depth of the Rolandic sulcus. Excision of the whole Area 6a, that is to say Areas 6aα and 6aβ, without interference with Area 4, has not been described in man.

Perhaps one of the most ingenious conceptions of Hughlings Jackson is his doctrine that the whole cortex of the brain, the highest levels as well as the middle levels, contain nervous arrangements representing movement and impressions, but unfortunately time will not permit me to deal with this subject.

In this lecture I have covered only a small part of neurology, yet I believe that I have demonstrated that in this field all the pioneer-work was done by Hughlings Jackson. The doctrines he developed have been, and will be for ever, a guide-post for subsequent research. But there is no other field of neurology with which Hughlings Jackson was not equally familiar. There is scarcely a single neurological problem which was not illuminated and elucidated by his prophetic ingenuity. Jackson's writings are the Bible of Neurology, the canon for the votaries of our science.

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THE INFLUENCE OF EXPERIMENTAL LESIONS OF THE SPINAL CORD UPON THE KNEE-JERK.

II—CHRONIC LESIONS.

WITH AN APPENDIX "A NOTE ON THE 'SPINAL' AND 'DECEREBRATE' TYPE OF KNEE-JERK IN THE CAT."

BY E. G. T. LIDDELL.

(From University Laboratory of Physiology, Oxford.)

USING the inhibitability of the knee-jerk as a criterion of "spinal shock," Fulton, Liddell and Rioch [9, 10, 11] showed that a vulnerable area of the spinal cord is the ventrolateral. They found that a lesion in that area was followed by increase in susceptibility of the jerk to inhibitory stimuli, which was well marked but somewhat less in degree than that produced by total section of the cord. And they concluded that severance of the descending vestibulo-spinal tract was the essential factor in lowering the resistance of the jerk to inhibitory stimuli [11, 12]. Similar results were obtained by a lesion of the medulla in the region of the vestibular nucleus [12].

Since their observations were made only after acute lesions of the cord, it became of interest to observe the state of "shock" some time after the lesion, or, in other words, to inquire into the progress of the spinal centres after withdrawal of the fringe of excitation from the vestibule and mid-brain, and to find out how soon recovery or compensation develops. Observations have already been published upon the recovery of isolated spinal centres after complete transection of the cord [19].

Preparatory operation.—The operations were performed with full aseptic precautions, Nembutal intraperitoneally being the anæsthetic. Small young cats were used, their weight averaging 2 to 3 kg. In all fifty preparations were made and examined. After laminectomy about the level of the last rib and exposure of two roots, the cord was laid bare more ventrally on one side by resecting bone, so that, when rotated slightly by holding the dura in mouse-tooth forceps at a point midway between the roots, the ventral spinal vessels came into view. Up to this stage care had been taken not to perforate the dura, especially dorsally, since in earlier experiments it was found that removal of the dura and the slightest injury of the dorsal roots gave rise to a massive

scar containing nerve-roots and muscle, with subsequent degenerations in the dorsal columns.

In order to avoid this gross scar in making ventrolateral lesions, the cord was gently rotated by holding the dura, as just described, until the ventral vessels became visible. A Graefe knife or fine scissors was then employed to pierce the dura latero-ventrally and sever the subjacent cord to the desired extent. It was not difficult to gauge the exact place and depth of the lesion, and, having the ventral vessel as a guide, to avoid transgressing the midline (fig. 1). No attempt was made to close the dural opening. The spinal muscles, deep fascia and skin were afterwards sutured in layers. Immediately after the operation and

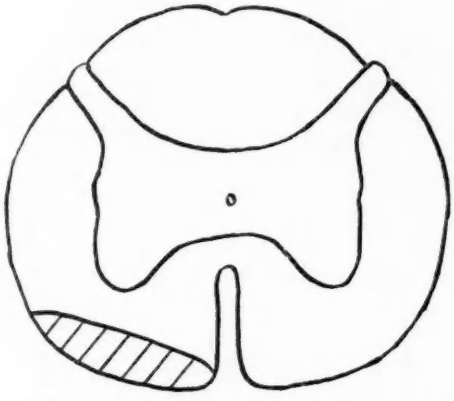


FIG. 1.—Diagram to show approximate area and extent of the ventrolateral lesions.

before recovery from the anæsthetic the knee-jerk on the side of the lesion was found to be slow in rise and fall, and the leg tended to swing back and forth after the fall. On the other side, the jerk was quicker, without subsequent oscillations.

Post-operative progress.—On the day following the operation and for some days subsequently it was common to observe weakness in both posterior limbs, together with distension of the bladder and overflow. This condition was probably due to transient œdema of the cord because, after a few days, the animal recovered and became able to walk, and about the same time the bladder began to empty normally. This post-operative paraplegia was more transient when the lesion was made by a knife rather than by scissors. Fulton and Keller have also preferred the ophthalmological knife to scissors [14]. When voluntary movement

is re-established and the animal held up by the scruff of the neck, the leg on the operated side is seen, at rest, to be limply dependent in an attitude of slight flexion. Although some patience is needed to examine such a non-co-operating subject as a cat with good voluntary power, it is possible to observe that to passive flexion imposed by the palm of the hand placed under the pad, the limb feels flaccid, or partly so, on the side of the lesion. But on the unoperated side the limb's posture is one of semiflexion, as in normal animals when held up by the neck, and the limb feels more resistant to passive flexion [29]. The knee-jerk on the operated side is more "spinal," that is to say, its rise and fall seem slow, but the movement is ample and the leg swings back and forth afterwards.

Later post-operative progress.—Within about two weeks of the operation an interesting change began to declare itself in the limb. In walking or running the limb on the side of the spinal lesion begins to show features of rigidity. These changes, which will be discussed more fully in a subsequent communication and are adverted to in the appendix to this paper, are due, it seems probable, to the development of a scar in the spinal cord which spreads dorsally and involves long paths from higher centres [25, 26].

Myographic examination.—The method of experiment was to decerebrate at the intercollicular level under profound ether anæsthesia, to allow recovery, and after immobilizing all muscles except the two vasto-crurei either by nerve section or tendon resection, to investigate with an isometric myograph the effect upon a series of knee-jerks of a single inhibitory induction shock applied to the ipsilateral sciatic nerve. As was shown originally by Ballif, Fulton and Liddell [2] resistance to inhibition is rapidly re-established in a decerebrate preparation and only gradually after total spinal section. As a result of this myographic analysis it is found that even some months after the spinal lesion, the jerk on the unoperated side resists a single inhibitory ipsilateral break shock as markedly as a normal decerebrate preparation. Contrasted with this, the jerk on the side of the lesion is more susceptible to inhibition, and for a longer period. The effect is profoundest soon after operation, but gradually decreases with the passage of time, until, after about a year, it is slight and the recovery curves on the two sides are similar to the curves from preparations decerebrated without any preceding spinal lesion. Figs. 2 to 4 with their legends give the essential data of the experiments. The figures show the speed of recovery of the knee-jerks in three typical preparations. As a rule, in the first hundred days after the lesion there is greater susceptibility to

inhibition on the one side. During the second hundred days recovery comes about. And in the third hundred days, heightened knee-jerks appear in the isometric records, while the regained resistance to inhibition continues, or even exceeds normal.

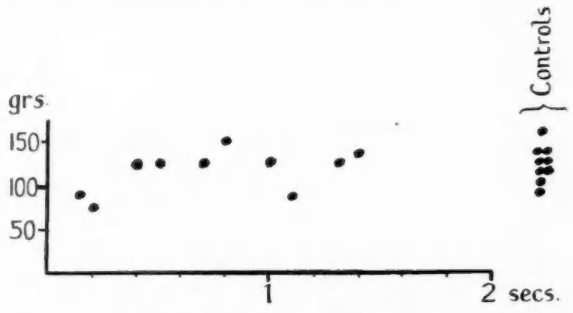


FIG. 2A.—Recovery curve of knee-jerk from inhibition on "normal" side, i.e. without spinal lesion. Inhibition produced by single break-shock to ipsilateral sciatic nerve. Isometric myograph. Intercollicular decerebration. Ordinates, tension of jerk in grammes. Abscissæ, time after break-shock in seconds. Control jerks shown on the extreme right. Extension of muscle 3·6 mm. (total resting length 1,100 mm.), to develop initial tension for experiment of 75 grm. Tendon taps, 600 grm. given by index finger. Tendon movement × 60. The jerks were small because the preparation was so rigid that occlusion took place.

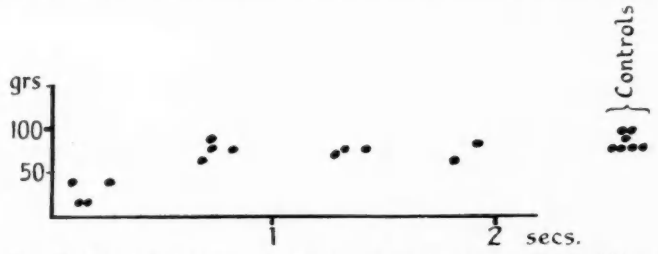


FIG. 2B.—Similar recovery curve from the same preparation on side of which a ventrolateral lesion was made in the cord thirty-nine days previously. Extension of muscle 6·4 mm. to develop initial tension of 80 grm. Taps 500 grm. There is more inhibition in this limb as compared with opposite normal limb, fig. 2A. Extension of muscle was adjusted to give jerks comparable in tension to the normal jerks shown in fig. 2A.

Another item of evidence regarding the "spinality" of the limb on the side of the lesion is found by observing the degree of extension needed to produce adequate backgrounds of excitation.

Number of days since lesion	Resting length of muscle in cm.	Extension in mm. necessary to give adequate initial tension on myograph	
		Side of lesion	Opposite side
39	11·3	6·4	3·6
98	—	1·8	2·0
105	11·9	3·6	1·9
125	10·7	2·5	2·1
138	11·5	5·9	1·3
350	11·5	1·4	3·6
388	—	3·2	6·3

In the table are given figures from a selection of preparations. For the first 100 or 150 days, the knee extensor on the side of the lesion has to be lengthened more by lowering the table in order to give adequate initial tension. In other words, to give the same "tonic" pull at rest,

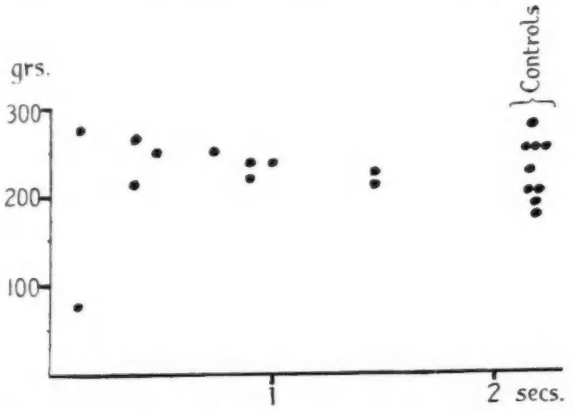


FIG. 3A.—Similar to fig. 2A, in preparation in which ventrolateral lesion was made in opposite side of cord 138 days previously. Extension of muscle 1·3 grm., to give initial tension of 90 grm. Length of muscle 1,150 mm. Taps 500 grm.

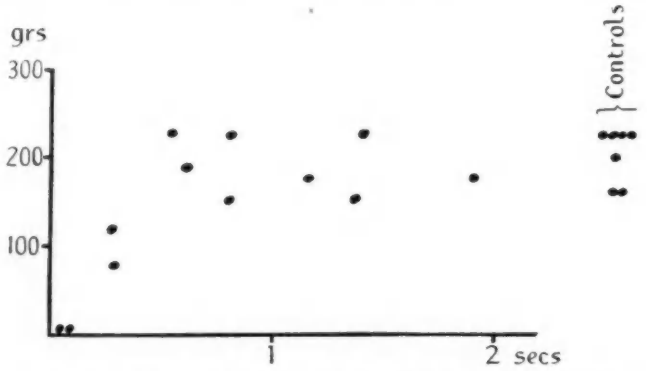


FIG. 3B.—Same preparation as fig. 3A, but on same side as the 138 days old lesion. There is greater inhibition on this side and the muscle needed 5·9 mm. extension to give initial tension of 115 grm. which was adequate for elicitation of a comparable jerk. Tendon taps 650 grm.

that knee extensor's stretch-organs must themselves provide a greater measure of C.E.S. in the spinal centres owing to the diminution of background C.E.S. brought about by severance of the vestibulo-spinal tract on that side. After 300 days it is apparent that the position is

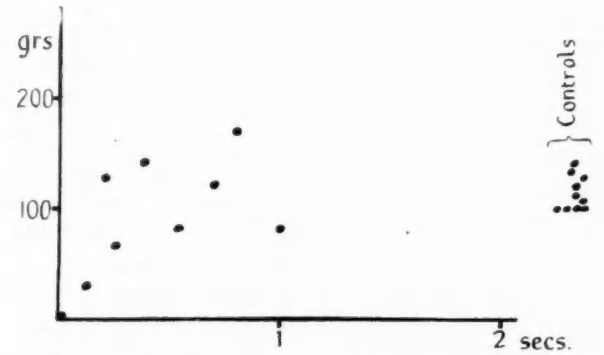


FIG. 4A.—Similar to figs. 2A and 3A, being the knee-jerk's inhibitory curve in preparation in which a ventrolateral lesion was made on opposite side of cord 350 days previously. Extension of muscle 3·6 mm. to give initial tension of 100 grm. Length of muscle 1,150 mm. Taps 175 grm. Although less resistant to inhibition than preparation in fig. 3A, the curve is within the limits of normality for a decerebrate preparation.

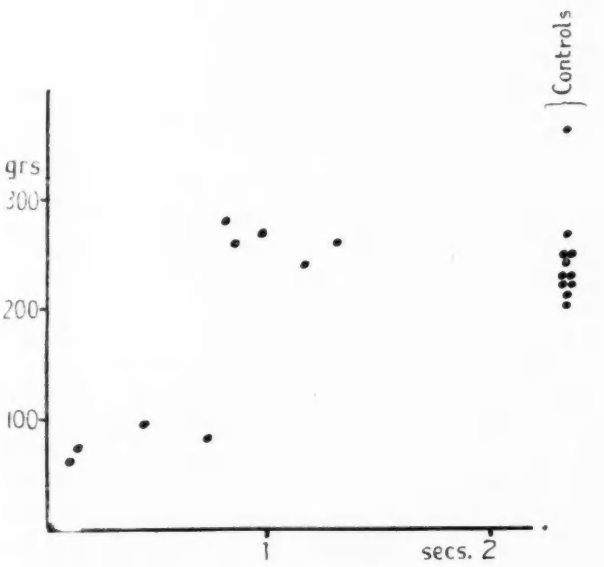


FIG. 4B.—Curve from side of lesion. The jerks are larger, although the extension of the muscle was less, 1·4 mm. Initial tension 50 grm. Taps 175 grm. "Overcompensation" has supervened as regards posture. Recovery curve from inhibition is similar to fig. 4A. In that respect also compensation established.

reversed and over-compensation, whatever its provenance or nature, has come into evidence. In the stage of over-compensation, the muscle on the side of the lesion needs less extension than the muscle on the "normal" side, in order to give the same initial tension, and that, be it noted (1), after ablation of all nervous structures headward of the inferior quadrigemina (2), in spite of, or because of, the spinal scar already mentioned. In all these preparations voluntary power had been excellent before decerebration. Histologically, the cord shows ipselateral degenerations in the ventro- and dorsolateral regions, but not in the dorsal columns.

In a number of preparations, spinal lesions were made laterally or dorsolaterally. Such preparations showed but little tendency to post-operative paraplegia or to distension and overflow of the bladder, presumably because the more accessible approach to the cord minimized operative trauma, slight though that was in the ventral approach. Ipsilateral limb rigidity [25] appeared on recovery from the operation, i.e. within two days, and it was of similar degree to that ensuing from the dorsally spreading scar after ventrolateral lesions, i.e. more than 1·0 kg. on the ipsilateral side, less than 0·5 kg. on the contralateral. When decerebrated at any time, the ensuing decerebrate rigidity was, as a rule, equal in the two hind limbs, or very slightly less on the ipsilateral side. There was no clear difference between the knee-jerks after decerebration, and increased inhibitability of the spinal centres was too slight to be detected, even if present. These findings, therefore, confirm the belief of Fulton, Liddell and Rioch, that, as concerns inhibitability of the knee-extensor's motor centres, the ventrolateral area of the cord is an area of special vulnerability, and lesions in it are followed by increased susceptibility to inhibition.

Conclusions.

(1) Lesions have been made in the ventrolateral area of the spinal cord of cats at the dorso-lumbar level. When the preparations are examined after intercollicular decerebration, there is found an increased susceptibility of the knee-jerk to ipsilateral inhibition.

(2) The susceptibility diminishes with time, and after the lapse of 100 days and before the lapse of 200 days, resistance to inhibition reaches normal value.

(3) The reflex resting tension of the knee-extensor also recovers gradually and approximately *pari passu* with increased resistance to inhibition.

(4) After 200 days there occurs over-compensation, both as regards inhibition and reflex resting tension.

(5) A sequel of ventrolateral lesions is a scar spreading dorsally (but not as far as the dorsal columns), whence arises a limb rigidity which is other than decerebrate rigidity and is itself abolished by intercollicular decerebration. The previously rigid limb then becomes flaccid. This flaccidity after decerebration is due to the original ventrolateral lesion.

I am indebted to the Christopher Welch Trust for a grant towards photographic expenses, to Mr. T. A. Marsland for microscopic preparations, to Mr. A. S. Chapman for his care in looking after the operated animals, and to Mr. C. Carr for his help with the experiments.

APPENDIX.

A Note on the "Spinal" and "Decerebrate" Type of Knee-jerk in the Cat.

In the preceding paper it was mentioned that a sequel of lesions in the ventrolateral region of the cord is a scar which spreads dorsally (but not so as to involve dorsal columns), producing rigidity in the ipsilateral limb within two or three weeks, presumably by involvement of paths from high nerve centres [3, 25]. The rigidity lasted for a year or more (as long as the observations were made), and from the end of the first month to the fourth or fifth was very evident in the gait. It came into prominence especially when the animal was held up by the scruff of the neck and the two sides compared by manual and dynamometer pressure on the pads of the hind feet. The animals became friendly and docile, and when their attention was distracted by petting, the limbs could be examined without interference from voluntary movements. It was common to find, in a cat weighing 2 to 3 kg., that a dynamometer pressure of 1·0 kg. was needed to flex the rigid limb, whereas 0·1 kg. was sufficient for the normal limb [18]. When the rigid limb had once been flexed, its return to extension was so greatly accelerated by gentle pressure on the pad of the foot that the Magnet reaction [31] appeared to be in operation. At all times voluntary movement, when elicited, was good, so that serious impairment of cortico-spinal paths was improbable, though the rigidity in the one limb often hampered great activity or friskiness, and for walking, required circumduction of the limb. A "clasp-knife" effect was not detected, and resistance to passive flexion was maintained until flexion was complete. On occasion, the ipsilateral limb was so rigidly extended that its knee-jerk was occluded. In the experiments now to be described, the animals were decerebrated at intercollicular level, so that long paths beginning (or ending) above that level were severed. The severance of the vestibulo-spinal tract of the cord achieved by the original lesion in the ventrolateral area remained with the same pattern of effect as before, though now no longer a palimpsest with higher nerve patterns.

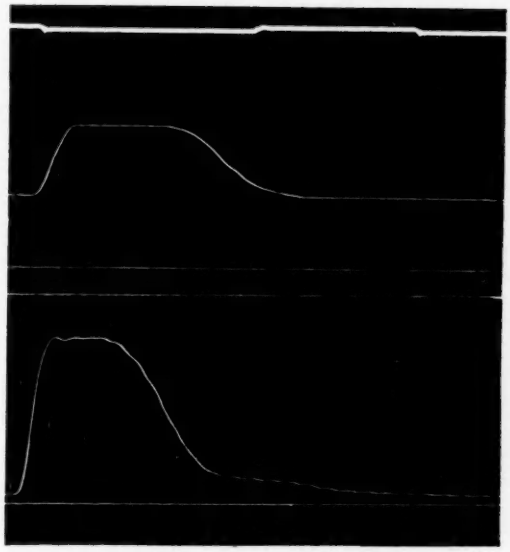
When such a preparation is examined after decerebration, it is found that on the unoperated "normal" side the limb shows all the well-known plastic characteristics, such as shortening and lengthening reactions and "clasp-knife" rigidity [24, 4]. But on the side of the lesion there is marked alteration. The limb on that side is now flaccid. Removal of nerve-tissues headward of the intercollicular level has, in fact, banished rigidity in that limb, and reveals the preparation in the familiar aspect of a decerebrate preparation which has suffered unilateral loss of the vestibulo-spinal tract. That decerebration at intercollicular level should abolish the previous rigidity shows, in brief, that it and decerebrate rigidity have neither a common provenance nor closely related source, but impress a somewhat similar pattern of effect on the lower reflex arc. Such a conclusion is more than anticipated by Sherrington's account of decerebrate rigidity when he found that unilateral extirpation of a cerebral hemisphere produced ipsilateral rigidity, but stimulation of the motor area in the remaining opposite hemisphere inhibited the rigidity [27]. Without gaining for the present much further insight into the origin of the rigidity previous to decerebration, it is appropriate to recall Hughlings Jackson's dictum on the rigidity of hemiplegia that negative states of nervous centres cannot cause positive states, though they may permit them. The established belief in the inhibitory action of the cerebral hemispheres on extensor tone and its appearance after cerebral lesions has received much experimental support [5, 14, 15, 16, 17, 22, 24, 30]. But removal by decerebration of such limb rigidity has not often been described in the literature. This problem will be discussed more fully in a later paper [20].

The clinical changes brought about by decerebration can also be effected qualitatively by moderate anæsthesia from nembutal (35 mgm./kg. *i.p.*) Nembutal, indeed, appears to produce in that respect a sort of pharmacological decerebration. The properties of dial are very similar [13]. During nembutal anæsthesia there is not, it is true, any great degree of decerebrate rigidity in the normal leg, but there is in it much more postural tonus (which is the same thing) than in the leg on the side of the spinal lesion, which is completely flaccid. The knee-jerks have the same qualitative characters as after surgical decerebration. Since the administration of nembutal has the advantage over surgical decerebration that recovery to the cerebrate state ensues in a few hours, periodical examinations under nembutal became customary, and the progress of the animal's condition was followed by its aid. At the close of the period of observation, that is after a period of from one to fifteen months, surgical decerebration brought confirmation of the reactions under nembutal. At the same time, it was demonstrated on every occasion that the difference between the jerks on the two sides was not due to any action of the flexor muscles, since the differences were not affected by paralysis of the flexors by nerve section.

Examination of the Knee-jerk.

When a preparation in which a ventrolateral lesion in the spinal cord has been made previously is examined in the supine position: (*a*) Under moderate

anæsthesia by nembutal; or (*b*) after surgical decerebration at intercollicular level, it is found that the knee-jerks are very different on the two sides. To elicit the jerk, a percussion hammer is used, of the National Hospital pattern, though with a rigid and not a whalebone handle. Such a hammer is, therefore, relative to the cat a large hammer, engaging, probably, all or most of the stretch-receptors in cat's muscle. In order to obtain graphic records a recording mechanism was employed which consisted of a bell-crank lever with a myograph arm of straw and a frontal writing point of parchment, writing upon a smoked drum. The animal was laid supine, in a rigid cradle, with the

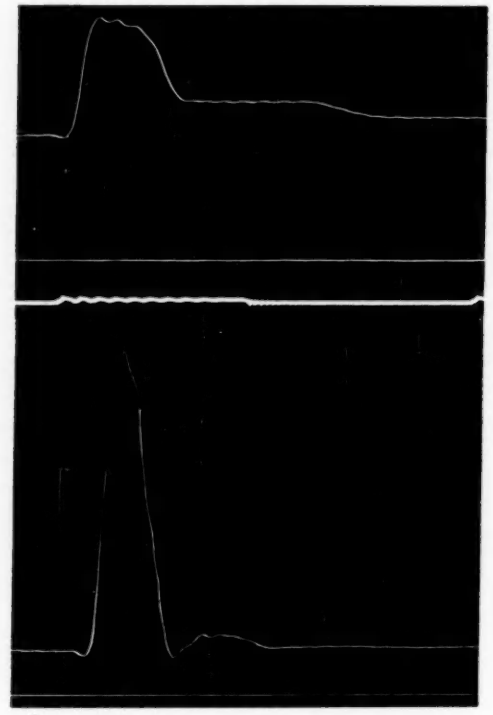


A.—Records of knee-jerks made with myograph which was mainly isotonic and allowed shortening, but was also isometric, through the restraint of a rubber band. Frontal writing point. Tendon movement multiplied four times. Top, A1, knee-jerk on normal side, preparation under nembutal. Bottom, A2, jerk on side of ventrolateral spinal lesion made 33 days previously. Difference in initial tension less than 10 grm. Time 0·6 secs.

thighs supported by a padded bar and the legs dependent. The animal's foot was encased in a light shoe which was attached by a thread to the vertical arm of the myograph. The lever of the myograph was restrained by an elastic band, so that the mechanism was neither strictly isotonic nor isometric. Tendon movement was magnified four times. Isometric examination, although it has yielded a wealth of information, is not conformable with most voluntary muscular contractions (except in " tug-of-war ") because it does not permit of shortening. Isotonic myographic examination, on the other hand, although conformably similar to most muscular activities has been a fertile source of

erroneous conclusions in which the mechanical properties of levers have been interpreted as physiological phenomena. But since it is an isotonic contraction which is observed in clinical examination of the knee-jerk, myographic recording which is mainly isotonic and only isometric to the degree of reducing instrumental error, seems to be the logical method.

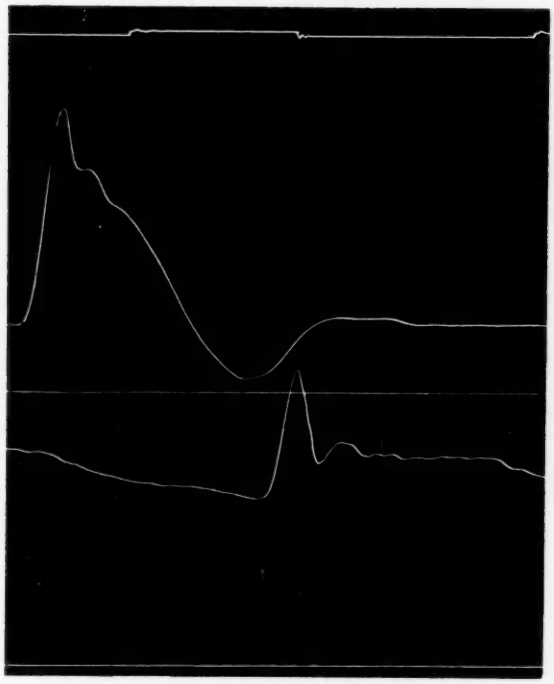
At myographic examination, confirming previous clinical examination, the knee-jerk on the side of the spinal lesion, especially in the first hundred days,



B.—Same preparation, five days later and three hours after decerebration at intercollicular level. Difference in character is maintained and accentuated. There is less posture on the side of the lesion (bottom record) and so much "fling" that the writing point fails to trace the upper part of the rapid upstroke. This preparation also provided the recovery curves of fig. 2 (isometric records) in preceding paper.

rises freely and abruptly, and then falls abruptly (figs. A2, B2), the limb oscillating two or three times after reaching the base-line (fig. B2). The oscillations are not so clear in the myograph records as in the free state of the limbs. On the normal side, however, the rise of the jerk is more gradual, its turn at the summit is more gradual as a rule, and its return to the base is gradual and not

followed by oscillations (figs. A1, B1). The difference between the jerks is reminiscent of the difference between a freely vibrating spring and a damped spring. Of the threshold value of the stimulus, it can be said that in the free or isotonic state adequate stimulation of the ipsilateral knee is apparently achieved by a lighter tendon tap, e.g., from a pencil, than is the contralateral (normal) knee. Under those conditions, however, the slight tap to the normal knee may be held to elicit such a small response that it is occluded by the strong postural



C.—Similar to B, 138 days after lesion in ventrolateral region and two and a half hours after decerebration at intercollicular level. Top record, C1, from side of lesion. Bottom record, C2, from normal side. Difference between initial tension of two records is 10 grm. The normal jerk is more peaked than usual, but at every level it is broader than on the operated side. These records show also that, in spite of partial occlusion of the jerk on the normal side : (i) the jerk on that side has greater breadth, (ii) the difference is evident as late as 138 days after the lesion, though much diminished. Posture has appeared on the operated side (top record).

background. Under strict isometric conditions, on the other hand, in the first seven months after the spinal lesion, a higher threshold is known to exist on the ipsilateral side, that is to say, a higher static initial tension must be applied to the tendon to produce an adequate background of C.E.S., and often, in addition, higher kinetic tension in the tap (figs. 1 to 4). The disparity, there-

fore, between the necessary tensions under isotonic and isometric conditions is more apparent than real.

The most noticeable feature in the isotonic record is the sharp steep rise of the jerk and its height, on the side of the lesion (fig. B2). In that phase of movement, inertia of the lever could not account for the steepness of the rise, but would rather tend to reduce it. Nearer the summit the inertia of the lever will induce errors, even though the lever is partly isometric in virtue of the rubber band, and in the present series that segment of the record does not merit too close analysis, though its general contrast with the restrained sweep of the normal knee-jerk is significant of the difference existing between the two sides. As a rule, the plateau of the jerk on the normal side (fig. B1) is longer than on the operated side. It is never shorter. Its length must be ascribed to "tonic after-discharge," since the other factor which could produce a longer plateau, that is increased initial tension, though here too slight, being only a few grammes (less than 10) might, if more considerable, and under more strictly isometric conditions, operate in favour of the other leg which is known, from isometric records, to need greater initial tension (see p. 164).

These differences are most marked in the first hundred days after the spinal lesion. Thereafter they diminish, but are easily demonstrated during or after the second hundred days, when there is some posture on the operated side (fig. C1 and C2).

Discussion.

Taking these features as a whole, one sees on the normal side how "posture follows movement like a shadow." On the operated side graduality of movement is lost by the absence of posture.

One feature of the jerk on the operated side cannot so readily be ascribed to the absence of posture. That feature is the rapid rise. Posture, if present-day beliefs are correct, maintains or tends to maintain an already existing tension in the muscle's tendon, but does not markedly affect the speed of contraction, though a small increase is theoretically possible as a result of sub-threshold fringe effect [4]. But in the operated limb there is an absence of posture, and that is the limb with the rapidly-rising jerk. Some other line of argument must therefore be sought. There may, for instance, be a more synchronous central discharge from the reflex centres when posture is absent, though it has not been observed by workers with electrical methods. But it occurs in flexor muscles, and there is ample evidence, myographic and electrical, of release after spinal transection [7, 28, 21]. For extensors, however, after spinal transection, the quicker white muscle is inactivated from shock, leaving only slow red muscle [6]. Alternatively, extensor's central discharge may have been slowed [23]. Now isotonic contractions of muscles, as is well-known, can be slowed by fatigue, and there ensues an increased height in the isotonic myogram, as in the staircase phenomenon. In the present series of experiments sections of certain spinal tracts has been made, so that in many of its reactions the muscle's behaviour is "spinal" in type.

Slowness of contraction, whatever its cause [2, 6, 23], is a feature of " spinal " behaviour. Hence the slow " spinal " muscle may endow the myograph lever with greater inertia so that it reaches a higher level. This explanation, however, is somewhat weakened by the fact that the lever is not purely isotonic and that its inertia is checked by the indiarubber band, which, when the lever is unattached, brings it rapidly to the base line. Another possibility is favoured when slow muscle is active. It has been noted by Fröhlich [8] and quoted thus by Adrian [1]: " increased duration of the contraction may lead to an actual increase in the tension exerted by the whole muscle, because the longer the duration the more chance there is that all parts of the muscle will be fully contracted at the same moment." This seems a very probable explanation of the higher contraction of the spinal knee-jerk.

Yet another possibility, however, deserves mention. In a tonic muscle, since some stretch-receptors appreciate tendon-tension and are active, so that some muscle fibres, as effectors in the reflex arc, are contracted, there is, thanks to the parallelism of fibres, protection of other tension-receptors from the tendon's pull, and they remain inactive. Hence when the tendon is tapped, these other tension-receptors are thrown into activity *seriatim*, that is, they are recruited, with the result that the reflex response of the tonic muscle is a gradual rise. The state of affairs in the atonic muscle is, however, far different. In such a muscle, if the stretch-receptors are active (and there is no reason to suppose that they are not), their excitation falls upon the group of central motoneurones in a state, as they are in these experiments, of partial though severe, spinal shock, i.e. with diminished low grade C.E.S., so that the motor centre remains unresponsive to scattered stimuli from the muscle. In consequence, the muscle is slack. In such a muscle, therefore, few or no stretch units are protected by the pre-existing activity of other muscle groups as they are in the tonic muscle, and all stretch units are equally susceptible to a tendon-tap, which, when it comes, throws all or nearly all stretch units into activity almost synchronously, and an almost synchronous volley of impulses travels to the reflex centre. The consequent reflex discharge is almost synchronous, or at least more synchronous than in the tonic leg, and an abruptly rising knee-jerk is the outcome. Hence may ensue the steep rise and fall in the almost apostural jerk, and the gently graduated (recruited) rise and fall in a more actively tonic muscle. Which of these arguments, favouring the theory of slow muscle or the theory of peripheral recruitment of stretch units, should be invoked in whole or in part, must remain an open question until electrical records have been made. Two other and subsidiary conditions which might affect the field of experiment deserve to be mentioned: (*a*) In postural muscles, occlusion tends to reduce the size of the tendon-jerk, but under the conditions of these experiments, differences in resting tension—only a few grammes—are too slight to produce substantial difference between the two sides. (*b*) These experiments were performed when some measure of recovery from spinal shock had come about. If a completely atonic muscle had been examined, there would, of course, have been no jerk because C.E.S. would be always of sub-threshold value.

Finally, there deserves to be mentioned autogenetic inhibition of muscle [4] which, if it is a reality in spite of some recent evidence to the contrary, may well suffer abeyance by severance of long ascending spinal paths, so that the "spinal" muscle is free to give a rapidly and high rising jerk, whereas the "decerebrate" muscle is not.

SUMMARY.

The mechanical differences between spinal and decerebrate knee-jerks have been examined with a rubber-checked isotonic myograph, and probable causes for these differences are discussed and analysed. Thus, any slow muscle gives an increased height of response with an isotonic lever, and "spinal" muscles are slow muscles. Also, in any apostural muscle it is probable that a tendon-tap promotes greater synchronicity of discharge than in a postural muscle, where recruitment of stretch organs is inevitable. Both these factors may contribute to the larger excursion of the "spinal" jerk.

With the passage of time, the differences become less sharply defined, but are detectable in all preparations for at least 200 days.

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THE ORIGIN OF THE POSTERIOR CEREBRAL ARTERY.[1]

BY D. J. WILLIAMS (MANCHESTER).

ABBIE [3] has recently described the comparative anatomy of the posterior cerebral and posterior communicating arteries. He has agreed with Shellshear, Stopford and others, who have affirmed from anatomical evidence that the posterior cerebral artery, although functionally a branch of the basilar artery, is morphologically one of the terminal branches of the internal carotid artery.

This paper advances evidence on this subject derived from a study of the perivascular innervation of the cerebral vessels.

METHOD.

Normal adult human brains were used. The great vessels comprising the Circle of Willis and its main branches were carefully dissected from the brain tissue, the adventitia and the perivascular structures being maintained as intact as possible. The vessels having been fixed in formol saline, were stained by Stöhr's modification of Schultz's silver impregnation method [6]. Stöhr's technique was still further modified. It was found that the original method caused extremely rapid precipitation of silver on the surface of the tissues; reduction was complete in a few seconds, and therefore could not be efficiently controlled. When the stock solution of hydroquinone was used in a solution of 1 : 180, optimum reduction occurred in about eight to ten minutes. After mounting the tissue it was found that reduction continued to occur slowly and caused staining of the mounting medium by diffusion. To obviate this the tissues were fixed in five per cent. sodium hyposulphite for seven to ten minutes, and were afterwards well washed in tap water. The specimens were then mounted in glycerine and were examined under a dissecting microscope. The perivascular nerve network was stained uniformly black, while the connective tissue of the tunica adventitia remained a golden brown shade.

RESULTS.

The plexuses on the vertebral arteries were readily distinguished by means of the dissecting microscope and they were followed cranially.

[1] Work carried out in the Departments of Anatomy and Surgery in the University of Manchester, during tenure of the Professor Tom Jones Memorial Fellowship in Surgery.

The sympathetic nerve fibres were traced with ease on to the basilar artery in four adult human brains. The plexus on this artery was simple in type and was less dense than that on the internal carotid artery, which formed an intricate network around the vessel. The vertebral plexus extended on to all the symmetrical branches of the basilar artery. In many instances solitary non-myelinated nerve fibres were traced on to the small pontine branches of the basilar artery. They could not, however, be followed into the substance of the pons.

At the upper extremity of the basilar artery, at the point of origin of the two posterior cerebral arteries, the perivascular innervation was scanty and of a very simple plexiform structure. The plexus divided and extended on to the adventitia of the vessels. The nerve fibres extended as far as the junction of the posterior communicating artery with the posterior cerebral artery. In no case did it extend along the posterior cerebral artery, nor did it continue on to the posterior communicating artery. Although the vertebral plexus ceased at this level, both of these arteries were liberally supplied with a perivascular nerve network (figs. 1 to 3).

The internal carotid plexus was then traced on to the middle cerebral artery. This plexus was, as has been stated, very dense. The nerve fibres extended along the middle cerebral artery, and could be seen on the tunica adventitia of its larger branches. They pursued a more simple and discrete course along these vessels than on the parent trunk. At the point of origin of the posterior communicating artery a very large division of the perivascular nerve fibres occurred. The plexus ran along the posterior communicating artery in the form of parallel bundles. The number of nerve fibres was greatly in excess of the usual perivascular innervation of so small a vessel. At the junction of the posterior communicating artery with the posterior cerebral artery this caudal flow of the carotid plexus swept on to the latter artery, and was continued along the artery until its terminal branches were reached (figs. 1 to 3).

In five of the eight specimens examined there was no apparent communication between the terminal nerve fibres of the vertebral plexus and the posterior flow of the carotid plexus. In three cases one fine twig appeared to join a nerve fibre from the posterior communicating artery (fig. 3). No other connection could be demonstrated between the two perivascular systems. Structurally they were quite discrete. The vertebral perivascular nerve plexus extended on to the basilar artery and on to all of its larger branches, including the commencement

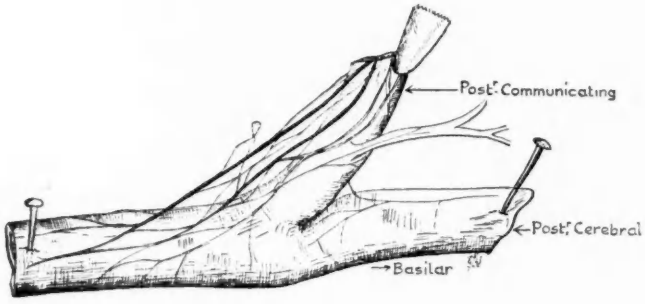

FIG. 1.

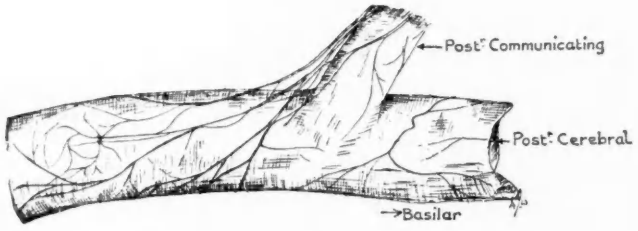

FIG. 2.

FIGS. 1 and 2.—Drawings of the junction of the posterior communicating artery with the posterior cerebral artery, stained to show the perivascular nerve network. Comparatively large nerve bundles are seen running from the posterior communicating artery on to the distal portion of the posterior cerebral artery. There they divide to form a typical perivascular plexus. Fine nerve fibres on the proximal part of the artery, derived from the vertebral plexus, have no connection with the carotid fibres.

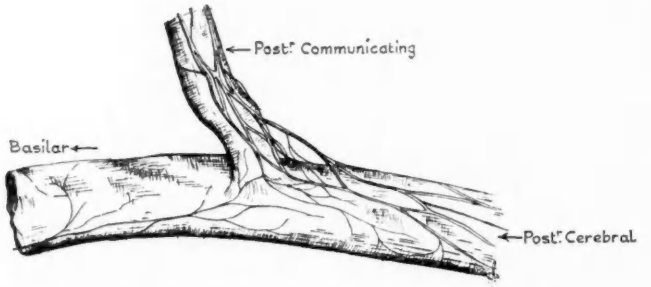

FIG. 3.—A similar specimen to those seen in figs. 1 and 2. Here a minute bundle of the vertebral nerve fibres is seen joining a bundle from the carotid system.

of the posterior cerebral artery. The carotid plexus innervated the anterior and middle cerebral arteries, and the posterior also, via the small posterior communicating artery.

Discussion.

This innervation of the posterior cerebral artery from the perivascular sympathetic plexus of the internal carotid artery is of phylogenetic interest. Abbie [2 and 3] recently described the comparative anatomy of the posterior cerebral artery. Briefly he showed that in amphibia (Rana) this artery was developed from the hippocampal artery of the lower forms. In reptiles (Sphenodon) it was mainly the hippocampal artery which had acquired a nearer source of origin for its blood, by means of a more posterior anastomosis with the cranial division of the internal carotid artery. In mammalia (Sheep) he found that the posterior communicating artery was larger than the posterior cerebral artery. In sub-primates the posterior communicating artery ran from the internal carotid artery to the basilar artery, and it was clearly the caudal division of the internal carotid artery. In primates the change is complete, and in man, except where developmental anomalies occur, the posterior cerebral arteries are apparently the terminal branches of the basilar artery.

Many observers have described wide variations in the anatomy of the posterior communicating artery. Stopford [7] found that it was greater than the posterior cerebral artery on both sides in 2 per cent. of cases and on either side in 4 per cent. of a series of cases that he examined. He described a case where, in the absence of a posterior communicating artery, the posterior cerebral artery arose as a continuation of the internal carotid artery, no anastomosis with the basilar artery being present. Fawcett and Blachford [4] found absence of the posterior communicating artery in 1·8 per cent. on the right, and in 1·4 per cent. on the left side. In these cases the posterior cerebral artery sprang directly from the internal carotid artery. Abbie [1] has described wide variations in the anastomosis between the anterior choroid, posterior communicating and posterior cerebral arteries.

During the present investigation the cerebral arterial tree of four early fœtuses was examined. In all the posterior communicating artery was relatively larger and thicker than in the adult, and its lumen was relatively wider. In one specimen, aged 9-10 weeks (fig. 4), it was equal in diameter to the posterior cerebral artery on one side, and greater than it on the other. Stopford [7] has stated that the

importance of the posterior communicating artery is much greater in intrauterine life, when it represents the origin of the posterior cerebral artery from the internal carotid artery. The perivascular innervation of the posterior cerebral artery which has been described in this paper is a striking confirmation of the views expressed by these previous workers. It is axiomatic that the nerve supply of any tissue follows that tissue from its ontogenetic point of origin. The posterior cerebral artery, deriving its whole innervation from the internal carotid system via the

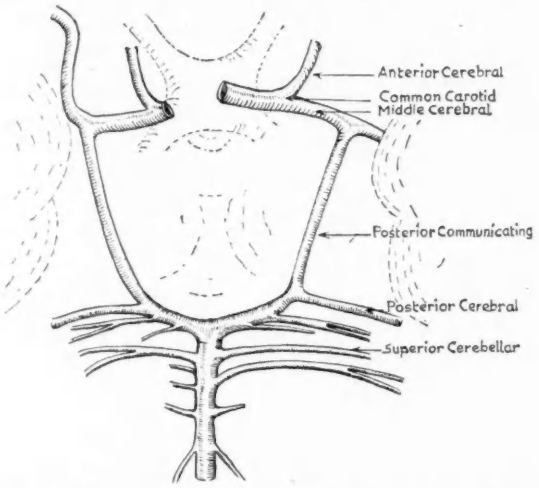

FIG. 4.—The Circle of Willis of a ten weeks' fœtus. The posterior communicating arteries compared with those of the adult, are much thicker and longer. In this specimen they are as wide as the anterior and middle cerebral arteries.

posterior communicating artery, would therefore appear to be morphologically a branch of the internal carotid artery. That is, all the cerebral arteries, anterior, middle and posterior, are fundamentally a complete system. Therefore that portion of the posterior cerebral artery between the basilar artery and the posterior communicating artery, and not the posterior communicating artery itself, is the true connecting link between the vertebral and carotid vascular systems.

Shellshear [5] has postulated that the distribution of arteries obeys some definite ontogenic and phylogenetic law. He has stated that the evolution of the vessels is dependent on, and proceeds hand in hand with, the evolution of their fields of distribution. Thus, as Abbie has said, the arteries of the brain, being as much a part of the neurovascular mechanism as the brain itself, have shown changes concurrent with the

more specialized development of the central nervous system. As the brain has expanded to meet new and increasing requirements, its arteries have extended to satisfy the increased functional demands. In the backward growth of the cerebral hemispheres over the mid-brain the distribution of the posterior cerebral arteries has been separated further and further from the parent trunk, the internal carotid artery. In accordance with natural economy a nearer course of supply has been found in the anastomosis of the basilar artery with the posterior cerebral artery. Concomitant with the increasing functional demands of the posterior cerebral artery, the basilar artery has enlarged adequately to supply the increasing amount of cerebral tissue. Although functionally the posterior cerebral artery is a terminal branch of the basilar artery, morphologically it is one of the three terminal branches of the internal carotid artery. The carotid system is therefore distributed to the whole of the fore- and mid-brains while the phylogenetically older vertebral system is strictly confined to the hind-brain.

SUMMARY.

In man, the development of the cerebral cortex has been such that the original distribution of the vessels can only be ascertained indirectly; first, by comparison with lower forms; secondly, by the variations from the normal picture; thirdly, by the condition in the early fœtus; and lastly, by a study of the perivascular nervous network.

In this paper the perivascular innervation of the posterior cerebral artery has been shown to be derived from the carotid sympathetic plexus. The condition of the vessels in the fœtus has been described and previous work on their morphology has been summarized.

All the evidence produced confirms the view that the posterior cerebral artery is morphologically a terminal branch of the internal carotid artery.

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MENINGITIS SEROSA CIRCUMSCRIPTA SPINALIS (SPINAL ARACHNOIDITIS).

BY J. ST. C. ELKINGTON.

In 1903, Spiller, Musser, and Martin [1] reported in some detail a case of spinal compression which was operated upon for a suspected spinal tumour and in which laminectomy revealed only a localized collection of fluid, under great tension, in the pia-arachnoid. No cause for this unusual accumulation of fluid was discovered. The patient recovered completely.

From this time similar cases are to be found in the literature, and in 1908 we find one reported by Mendel and Adler [3] under the title of "Meningitis Serosa Spinalis."

In discussing, in a subsequent paper, the nature of idiopathic collections of fluid in the subarachnoid space, Spiller [5] stated that practically nothing was known of their ætiology, and it was not until 1909 that the first attempt to define and describe this clinical entity was made by Sir Victor Horsley [6] under the title of "Chronic Spinal Meningitis."

He reported two cases in detail, and stated that he had operated on twenty-one in all. He regarded the condition as more common than spinal tumour, for which it was usually mistaken before operation. The essential change was a localized collection in the spinal subarachnoid space of fluid under great pressure, with secondary changes in the spinal cord which was often shrunken and on microscopy showed "meningogliosis." All Horsley's cases occurred in adults and he was inclined to attribute them to syphilis and gonorrhœa, stating his opinion that trauma played no part in their causation.

In spite of Horsley's paper the ætiology and clinical features of the condition remained obscure, and in 1910 Munro [9], in describing a number of cases, wrote "there is a something that produces a grave paralysis which is demonstrated clinically which appears totally inadequate and for which we have no corresponding experimental or post-mortem explanation."

From this time more numerous contributions to the subject appear in the literature under various titles, namely "Meningitis Circum-

scripta Spinalis," "Meningitis Serosa Spinalis," and "Chronic Spinal Arachnoiditis," and they fall naturally into two groups.

The majority of papers are simple case records with little or no further comment. Of these may be mentioned those of Bruns [2], Mendel and Adler [3], Bliss [4], Mills [7], Weisenburg and Müller [8], Kennedy [10], Bouché [11], Skoog [12], Batten [13], Marburg [14], Bausamer [16], Moniz [17], Barré [18], Grinker [19], Hiller [20], Kron [21], Hassin and Andrews [23], Guillain [25].

The minority of contributions present a series of cases with details of pathological investigations, operative findings and in some cases post-mortem material, and attempt to define the clinical features and ætiology of the condition. Of these the most noteworthy are the papers of Stookey [22], Mauss and Krüger [15], Vincent [24], Paulian and Turnesco [29].

Post-mortem material is unfortunately scanty, and detailed descriptions of the pathological findings are confined to the papers of Grinker and Marinesco. These contributions will be further referred to in separate sections of the following.

The present paper consists of an analysis of the symptoms and signs observed in forty-one cases of meningitis serosa circumscripta spinalis.

Only three of these were under the direct observation of the writer. The remainder were all under the care of past or present members of the staff of the National Hospital, Queen Square, and to them the writer expresses his gratitude for their permission to make use of the case notes. He is particularly indebted to Dr. Gordon Holmes for his unfailing advice and interest in the preparation of the paper and to Dr. J. G. Greenfield for his assistance in examining the pathological material and for the records of cerebrospinal fluid examinations.

In all cases there was reasonable confirmation of the diagnosis by laminectomy and in some cases by autopsy. With four exceptions the operations were performed by the late Sir Percy Sargent, and his private notes have been available for study and provide a valuable description of the operative findings. Owing to their length and number it is impossible to publish the clinical protocols of the cases.

Although in the recent literature on the subject the term "chronic spinal arachnoiditis" is more often employed, in the present contribution the title, "Meningitis Serosa Circumscripta Spinalis," will be retained as it does not suggest a limitation of the pathological process exclusively to the arachnoid.

Ætiology.—Analysis of the cases throws some light upon the dis-

tribution and ætiology of the condition. The disorder is distinctly commoner in men than in women. Of forty-one cases, thirty occurred in males and eleven in females. It might be supposed that this preponderance of male cases was due to the greater frequency of trauma and venereal infection in this sex, but in the group of cases in which no predisposing factor could be traced (eighteen) the same disproportion was observed, i.e. there were thirteen male and five female cases.

Although Hiller has reported a case occurring in a boy of 8 years, writers are agreed that the disease is predominantly one of middle life. This is confirmed by the present series in which the youngest case came under observation at 23, the oldest at 65; the average age of the patients when first seen was 45·1 years. Arranged in age-periods the forty-one cases are grouped as follows:—

Under 20 years	..	..	..	..	0 cases
20 to 30 ,,	..	..	..	..	6 ,,
30 ,, 40 ,,	..	..	..	..	6 ,,
40 ,, 50 ,,	..	..	..	..	14 ,,
50 ,, 60 ,,	..	..	..	..	10 ,,
60 ,, 70 ,,	..	..	..	..	5 ,,

Many different views have been put forward as to the cause of meningitis serosa circumscripta. Horsley suggested that syphilis and gonorrhœa were responsible and that trauma played no part, while Mauss and Krüger report in detail fifty-four cases following war injuries in which the traumatic origin appears indisputable. Others have regarded it as the consequence of various systemic infections, and Bouché specifically mentions typhoid. An association with spinal tuberculosis and arthritis of the spine is mentioned by Vincent. Several cases have been recorded following meningococcal meningitis (Batten, Holbaum, Guillain). The case reported by Kron followed a septic abortion.

Analysis of the present material amply confirms the inference that there are various conditions capable of initiating a similar process in the spinal meninges, and that there is, in fact, no single cause of the disease.

Of forty-one cases it was impossible in eighteen to trace any previous illness or injury which might have been the cause of the spinal condition. In many of these the usual illnesses of childhood were recorded; in one glycosuria was present; and in two a carbuncle had occurred some years previously.

In the remaining twenty-three cases, however, one or more possible causal factors were present, as follows:—

Trauma.—In nine cases a history of spinal trauma was obtained.

The interval between the injury and the onset of cord symptoms varied widely, being as short as three weeks in one case and as long as twenty-four years in another. The severity of the injury was equally inconstant.

Case 16 fell some distance on to her feet, and although the only symptom at the time was vomiting, radiograms taken eighteen months later showed a compression fracture of the 3rd and 4th dorsal vertebræ.

Case 18 suffered two injuries: on the first occasion a fall from a horse led to six months incapacity from numbness and weakness of the left arm, and twenty years later the patient was blown up and struck between the shoulders by a sandbag. At the time of examination radiograms showed an old fracture of the 5th dorsal vertebra.

Case 37 injured the cervical spine in a hunting accident and for four weeks experienced paræsthesiæ in the hands on flexing the neck. Radiograms taken ten years later showed an old fracture of the 4th and 5th cervical vertebræ.

Cases 25, 27, 38 suffered injuries to the spine followed immediately by weakness and paræsthesiæ in the limbs which cleared up completely to be followed by a variable latent period before the recurrence of symptoms. In them no evidence of actual fracture was obtained.

Cases 29, 39, 41 experienced no nervous symptoms at the time of the spinal trauma, and no radiological evidence of bony injury was obtained at the time of examination.

In the three cases in which radiograms showed an old fracture, the meningeal changes were confined to the neighbourhood of the damaged vertebræ.

Of the nine cases in which a history of trauma was obtained no other probable ætiological factors could be found in six, but in three cases a history of some form of infection was also present.

Infection.—A history of severe infection in the past was obtained in eight cases. The most convincing evidence that this was responsible for the subsequent meningitis circumscripta was found in Cases 19 and 33, both of whom were young adults who had suffered from proved meningococcal meningitis within two years of the onset of spinal symptoms. In both of these gross arachnoidal adhesions and loculation were found at operation.

In four cases a history of enteric fever from fifteen to thirty years previously was obtained, and in two pneumonia had occurred within two years of the onset of symptoms, but proof of any connection between the infection and the spinal disease is lacking.

Syphilis.—A history of syphilis was obtained in four male cases, the primary infection occurring two years (Case 1), ten years (Case 15), fourteen years (Case 8), and thirty-five years (Case 34)

respectively before the development of spinal symptoms. In two cases (1 and 8) no serological investigations were carried out. In Case 15 the earliest symptoms were those of an acute myelitis diagnosed at the time as syphilitic, and when further spinal symptoms developed after an interval of seven years the cerebrospinal fluid showed a lymphocytosis and a weakly positive Wassermann reaction. At the time of operation the cerebrospinal fluid was normal in all respects and the blood Wassermann reaction negative.

Case 34 acquired the primary infection at 20, developed gummatous ulcers of the legs at 27, and spinal symptoms at 52. The Wassermann reaction in blood and cerebrospinal fluid was repeatedly negative, and the latter normal except for slight increase in protein.

It may be questioned whether it is justifiable to include in this series cases in which definite evidence of syphilis was obtained in view of the widespread changes in the cord and its membranes so frequently caused by this disease. But the symptoms and operation findings did not differ from those present in the cases where there was no such history, and even in Case 15, where the patient never made a complete recovery from an early attack of myelitis, there was no serological evidence of active meningeal or vascular syphilis. It seems reasonable to regard syphilis as one of the several conditions capable of giving rise to a low grade inflammatory process in the spinal meninges which in its turn produces the mechanical disturbances of the subarachnoid space which characterize meningitis circumscripta whatsoever its ultimate cause.

Gonorrhœa, to which Horsley attributed some influence in the causation of this disease, had occurred in the past in four male cases without any evidence of a concurrent syphilitic infection. In none of these was there any evidence of the latter disease serologically.

In brief, in forty-one cases of meningitis serosa circumscripta a history of spinal injury was obtained in nine cases, of syphilis in four cases, of meningococcal meningitis in two cases, of gonorrhœa in four cases and of other systemic infections in six cases. In eighteen cases (44 per cent.) no cause whatsoever could be traced.

Clinical Features.

As might be expected the clinical picture presented by the disease is very variable. In the first place any part of the spinal cord or cauda equina may be affected; in the second place the process may be strictly localized or diffuse; and finally the onset may be rapid or very gradual.

Although in my cases it was often impossible to assign the

symptoms to disease of a single spinal segment we can estimate the site of maximal damage by the level taken to be the centre of the operative field at laminectomy and on this standard it is clear that no portion of the spinal cord is immune to the disorder.

The distribution was as follows:—

High Cervical (C.1-C.4)	6 cases
Cervical enlargement (C.5-C.8)	6 ,,
Upper dorsal (D.1-D.6)	15 ,,
Lower dorsal (D.7-L.2)	9 ,,
Lumbo-sacral enlargement and cauda equina	5 ,,

In discussing the length of history Stookey has stated that the disease has a very gradual onset, and 50 per cent. of his cases gave a history of more than nine years. This is not true of the present series for only three gave a history of more than five years, and of the forty-one cases 53 per cent. had a duration of less than one year and 85 per cent. of less than three years.

In spite of the differences of site, extent, and duration of symptoms analysis shows a considerable background of similarity in my cases.

Two modes of onset of symptoms were commonly encountered, viz. (*a*) spontaneous pain, and (*b*) symptoms of spinal compression, namely weakness or sensory disturbance below a certain segmental level.

Spontaneous pain was the first symptom in twelve cases. The pain was often sudden in onset, and variously described as "stinging," "burning," "gnawing," or "neuritis like." In some cases it was described as "sciatica" or "lumbago." The pain usually began on one side of the body, but later became bilateral. At first it was commonly confined to the cutaneous distribution of one or two spinal segments, but it usually spread until a wide cutaneous area was involved, differing in this respect from the root pain experienced in spinal compression from other causes.

The pain was aggravated by movements of the spine, and in many cases by straining, coughing, and sneezing. Horsley and Mauss and Krüger also comment on the occurrence of diffuse pain.

In twenty-nine cases the first symptoms were those of spinal compression such as might occur in any compressive lesion, and their precise nature was largely dependent upon the part of the cord involved.

In seventeen cases motor symptoms were the first to occur, manifesting themselves by dragging of the legs, weakness of the arms, muscular rigidity, or involuntary spasms.

In eleven cases disturbance of sensation occurred before any motor symptoms, showing itself by numbness, coldness and paræsthesiæ in

eight cases, and by clumsiness and inco-ordination in three cases. In one case the first symptom was defective control of the bladder.

Whatever the mode of onset, whether with spontaneous pain or symptoms of compression of the cord, whether rapid or gradual, examination of the patient usually showed well-defined motor, sensory and reflex signs of local disease of the spinal cord and its nerve-roots.

Motor signs.—In no less than thirty-four cases a spastic paraplegia with characteristic changes in the reflexes was found. In twenty-three the upper limbs were quite normal, but in nine there was atrophy and hypotonia of the muscles of the hands and arms, and in two the upper extremities shared the spastic weakness present in the legs.

Of the seven cases in which a spastic weakness of the lower limbs was not present, five cases showed signs of disorder of the lumbosacral enlargement or of the cauda equina, i.e. weakness and wasting of the corresponding muscle groups with diminution or loss of their tendon reflexes.

In two cases motor signs were absent, and the reflexes were normal.

Sensory disturbances.—The commonest type of sensory disturbance was a diminution or loss of cutaneous sensibility below a certain segmental level. This was present in thirty-four cases. In twenty cases this disturbance was bilaterally symmetrical, but in fourteen one side of the body was more affected than the other. The upper level of sensory loss was well defined and identical for all forms of sensation in twenty-three cases, but in eleven cases the level was more irregular, and the sensory disturbance was dissociated. In eight of these cases the loss was more extensive for pain and temperature than for light touch, but in three cases the converse was true.

Cutaneous hyperæsthesia was present in six cases; the hyperæsthetic area was situated immediately above the upper level of sensory loss in three cases, and immediately below the area of chief sensory disturbance in three, and in two cases it corresponded accurately with the area of spontaneous pain.

In four of the cases in which there was not a general diminution of cutaneous sensibility below a certain level, patchy areas of sensory loss were present. They varied in extent, but in all cases the abnormal areas covered the cutaneous field of a number of adjacent spinal segments and were symmetrical, with zones of normal sensibility above and below. In all cases these bands of anæsthesia affected all forms of cutaneous sensation alike and were severe in degree, suggesting that they were due to involvement of the posterior spinal nerve-roots, in

contrast to the more uniform loss below a certain level which was found when the changes in sensation were due to medullary lesions. In three cases only was cutaneous sensory disturbance entirely absent.

Deep sensibility, like cutaneous sensibility, was frequently abnormal. The state of postural sensibility and sense of passive movement at joints is recorded in thirty-six cases, and in twenty-one it was disturbed in various degrees. With one exception all these cases showed also a gross loss of cutaneous sensibility in the affected parts. On the other hand fifteen cases presented no defect of postural sensibility in spite of the fact that in all except three of them considerable cutaneous sensory loss was present.

It appears, therefore, that the sense of position and passive movement, as ordinarily tested, are frequently normal in the presence of marked cutaneous sensory loss, but that the reverse is rare.

The sense of vibration at bony points is recorded in twenty cases. In five cases it was absent below a particular level, and in four of these the loss was associated with grave disturbance of postural sensibility, while in one the latter was normal. In eight cases the perception of vibration was diminished but not lost, and in all of these postural sensibility was defective in the same areas. In seven cases the appreciation of vibration was normal, and in all of these postural sensibility was also unaffected, although in some cutaneous sensory loss was present. These observations suggest that vibratory loss is associated with loss of sense of position and is unlikely to appear as an isolated phenomenon.

Function of the cranial nerves.—The state of the cranial nerves was recorded in thirty-nine cases, and was normal in thirty. In four cases slight pupillary abnormalities were noted. In Cases 3 and 6 this consisted of slight inequality in size without any other departure from normal. In Case 10 the pupils were unequal in size and the left was irregular in outline and reacted sluggishly to light. There was no other evidence of syphilis. In Case 34, who gave a history of syphilitic infection, one pupil reacted poorly to light. In five there was a slight degree of nystagmus. This was never sustained and was usually described as "slight nystagmoid jerks on full lateral deviation of the eyes." In one case mild signs of ocular sympathetic weakness were present on one side.

Disturbance of the sphincters.—The state of the organic reflexes is recorded in all cases. In fourteen they were normal, in spite of the fact that many of the patients had moderately severe paraparesis. In twenty-four cases early sphincter disturbance was present, showing itself

variously as a delay in starting, precipitance, or occasional incontinence. In only three cases was loss of sphincter control a marked feature, and in all of them a severe paraplegia was present. The sphincter disturbance is thus mild when compared with the severity of the motor and sensory changes.

Trophic changes.—Cutaneous lesions and bed sores were strikingly rare, and were only noted in three cases. In one the marks of a bottle burn were present, and in two there was evidence of old bed sores at pressure points. Horsley noted the late appearance of sphincter and trophic disturbances in this condition.

Vertebral column.—A record of the clinical examination of the spine was obtained in thirty-seven cases. In ten cases some abnormality was detected clinically, of which tenderness on pressure and percussion of the vertebral spines in the affected area was the commonest and was present in six cases. In five cases spinal rigidity was described. X-ray examination in those cases where there was clinical evidence of spinal abnormality showed arthritis in three cases, an old vertebral fracture in one case and diminution of an intervertebral space in another. In twenty-seven cases clinical examination revealed no evidence of spinal abnormality, but in spite of this radiograms showed an old fracture in one case, spinal arthritis in another, and doubtful erosion of one vertebra in a third.

Pathological Investigations.

The Wassermann reaction of the blood was determined in twenty-seven cases and was completely negative in twenty-six. These cases included four with a history of syphilis and four with one of gonorrhœa. In one case (Case 13) it was weakly positive, although no history or other signs of syphilis were present.

Cerebrospinal fluid.—Changes in the cerebrospinal fluid in cases of meningitis circumscripta spinalis have been described by several writers, but usually in reports of isolated cases. Stookey, in his series of thirteen cases, found manometric evidence of a complete subarachnoid block in six cases and of a partial one in four cases. In none was xanthochromia present.

On the other hand a complete Froin's syndrome was present in the cases described by Grinker, Hiller, and Guillain, and in one of Vincent's cases. In ten of Stookey's cases the cell-count was normal, but in three a pleocytosis was present, the cell-counts being 14, 12 and 6 lymphocytes respectively. Other authors have found moderate increase in the number of cells; Kron's case had 72 cells per c.mm., Vincent's cases 20 and 9

cells per c.mm. respectively, and that of Skoog 9 per c.mm. The majority of case reports in the literature, however, are in agreement in the absence of any increase of cells in the spinal fluid. All writers have found the Wassermann reaction in the cerebrospinal fluid uniformly negative.

In the present series the cerebrospinal fluid was examined in twenty-six cases although in some of the earlier ones the analysis was incomplete as judged by modern standards. In three cases fluid was unobtainable, and in twelve no attempt was made to obtain it. Manometric examination was carried out in only six of the forty-one cases. A complete block was present in one case in which the protein content was 0·11 per cent. A partial block was found in two cases with protein contents of 0·08 and 0·16 respectively. In three cases the pressure relationships as measured by the manometer were normal.

The cells were enumerated in each of the twenty-six cases in which the spinal fluid was obtained—and with one exception the count was normal (less than 5 per c.mm.). In one case counts of 50 and 42 lymphocytes per c.mm. were obtained on successive occasions, although there was no history of syphilis and the Wassermann reaction was negative in blood and fluid.

The total protein content was estimated in twenty-one cases and was normal (less than 0·05 per cent.) in eleven of them. In four cases it was slightly raised (0·05 to 0·08 per cent.), and in five cases there was a gross excess of protein (0·14, 0·2, 0·85, 0·11, 0·16 per cent.). The cases with great excess of protein all showed other evidence of loculation (i.e. yellow coloration, a positive Queckenstedt's phenomenon or arrest of lipiodol). Qualitative tests for globulin showed an increase proportional to the excess of total protein. It seems, therefore, that in meningitis serosa circumscripta marked increase in protein only occurs as a result of occlusion of the subarachnoid space. This is in agreement with the observations of Vincent and Stookey.

The Wassermann reaction was examined in the cerebrospinal fluid in twenty-five cases and in all it was negative. One case (Case 15) had given a weakly positive reaction on a previous occasion, but this was not confirmed at the time of examination.

The Lange colloidal gold sol reaction was carried out in thirteen cases and was normal (no rise above 2 in any dilution) in twelve. In one case in which there was a total protein content of 0·2 per cent. a meningitic curve was obtained.

Lipiodol examination.—Attention has been drawn by Barré, Hiller,

Moniz, Marinesco, and Paulian and Turnesco to the striking features presented in radiograms of the spine in cases of meningitis circumscripta after the introduction of lipiodol into the cisterna magna.

While in cases of spinal compression from other causes the opaque oil is held up in its entirety at the level of the spinal lesion, these authors find that in the presence of diffuse arachnoidal adhesions the lipiodol is arrested in droplets over a considerable extent above the site of the main lesion, giving an appearance reminiscent of a guttering candle. At the same time the arrest is incomplete and some of the oil is seen in the sacral end of the subarachnoid space.

The illustrations in the monograph of Paulian and Turnesco show this patchy arrest very clearly, but this appearance is by no means always present in otherwise characteristic cases. In cases reported by Guillain and by Vincent the whole of the opaque oil was arrested in one mass as in cases of tumour. On the other hand the writer has seen a widespread arrest of lipiodol in droplets of varying size in a case of diffuse vascular tumour of the cord.

In the present series lipiodol was introduced into the cisterna magna in seven cases only. In five it was arrested at a single level, which in each case was found at operation to be the site of marked abnormality of the subarachnoid space. In two cases the oil descended normally to the bottom of the subarachnoid space. The diffuse arrest referred to in the literature was not seen.

Diagnosis.

Spiller in 1909 and Horsley in the same year stated that meningitis serosa circumscripta spinalis resembled closely in its symptomatology spinal compression from tumour, and that it was usually diagnosed as such before operation. Careful examination of the present series amply confirms this statement, for the correct diagnosis was seldom suggested before operation, and even more rarely put forward with any degree of certainty. In spite of the improvement in accessory investigations it is probably equally true to-day.

Indeed it is difficult to see how a certain diagnosis of meningitis serosa circumscripta spinalis can be made before operation, particularly when one remembers how frequently spinal tumours, especially vascular ones, are associated with arachnoidal adhesions above and below the lesion. There are, however, certain features which should raise the possibility of this diagnosis in the clinician's mind when faced with a probable case of spinal compression. The disease as has been shown

is predominantly one of middle age and a history of definite spinal trauma, of past infection of the subarachnoid space or of syphilis is of value.

Though the more common mode of onset is that of an insidious spinal compression the commencement with spontaneous pain, when it occurs, is highly suggestive. This pain may be of sudden onset and is usually intense and intractable, and is diffuse in its distribution and frequently associated with hyperæsthesia. On examination the patient usually shows signs of a localized spinal compression together with a more diffuse involvement of the spinal nerve-roots. The root symptoms tend to be more conspicuous than in spinal tumours, whereas the spinal compression is slow in reaching an extreme degree so that trophic changes and disturbance of the sphincters are long delayed.

Unfortunately examination of the cerebrospinal fluid does not serve to distinguish the condition from spinal compression due to other causes. The value of lipiodol examination, as has been said, is stressed by French authors and certainly the diffuse patchy arrest of the oil over a wide area of the subarachnoid space when it is seen is highly suggestive of widespread adhesions in the subarachnoid space. This appearance may however be absent in cases of meningitis circumscripta and may be present in other conditions. It will thus be seen that while there are many points of value in the differential diagnosis between meningitis serosa circumscripta and other forms of spinal compression, there are no absolute diagnostic criteria.

It is true to say that the diagnosis can only be suspected and not established before operation.

Pathology.

The nature of the lesion present in cases of meningitis serosa circumscripta may be studied both from the findings at operation and by the more searching methods of biopsy and post-mortem examination. The former source is comparatively plentiful but the latter is unfortunately scanty.

Previous writers are in general agreement that the most conspicuous abnormalities seen at operation are to be found in the arachnoid. This membrane is unusually thick and often opaque in appearance. It is bound to the dura and to the cord and nerve-roots by adhesions which vary greatly in their number and extent. They may be localized or diffuse and often interfere with the normal circulation of the spinal fluid so that cysts are formed. In extreme cases they may be so extensive as to obliterate altogether the subarachnoid space.

Many authors draw attention to the great tension under which the fluid is contained within the arachnoid cysts and refer to the distended appearance of the dura and its feeble pulsation. The appearances of the arachnoid are admirably portrayed in a series of coloured plates in the paper of Mauss and Krüger.

The changes seen at operation, however, are not always confined to the arachnoid. In some cases the dura is thickened, and in the traumatic cases of Mauss and Krüger the epidural space contained organizing blood-clot and the epidural fat was undergoing fibrosis. Again changes in the spinal cord and its nerve-roots are frequently mentioned. Horsley was particularly impressed by the shrunken appearance of the cord. Skoog noted compression of the cord as if by an extramedullary tumour, and Bliss refers to changes in the pial circulation.

The present material confirms the previous findings and is of particular value as all the cases (forty-one) were submitted to laminectomy and with four exceptions the operations were performed by the same surgeon. In many cases his notes made immediately after the operation are available and are at times quoted verbatim.

Changes in the superficial tissues and spinal laminæ were noted in only five cases. In three, bony thickening was found at the site of an old fracture; in one case œdema, and in another fibrosis of the spinal muscles was present. In many cases (sixteen) the dura mater was recorded as being normal in appearance, but in some (eleven) it was noticed that the normal pulsations were greatly diminished or absent; in five cases the dura was found to be abnormally thick, and in some it was unusually tense on palpation.

It was on opening the dura, however, that the most striking and constant changes were seen. In eleven cases there were abnormal adhesions binding the inner surface of the dura to the arachnoid. The arachnoid itself was often milky and opaque in appearance, and in some the thickening had a patchy distribution. Of Case 3 it is noted that "the posterior surface of the cord showed alternate grey and white patches and there were small opaque areas in the pia like minute gummata," and of Case 8 that "the arachnoid was greatly thickened and appeared like flakes of lymph in peritonitis."

The most constant finding was the presence of abnormal adhesions between the arachnoid and the pia with consequent disturbance of the subarachnoid space. These adhesions were specifically noted in twenty-seven cases. They varied greatly in number and extent. In some they were strictly localized as in Case 15, where "at the 6th dorsal segment

there was an incomplete septum between the arachnoid and the posterior surface of the cord," and in Case 35 in which "a firm band of opaque arachnoid extended up and down for ¾ inch, constricting the cord at the level of the 4th cervical lamina." In other cases the changes were spread over a considerable length of the cord.

In association with the arachnoidal adhesions there occurred in eighteen cases loculated collections of fluid amounting to actual cysts. In four cases these were single, in two they were multiple, while in the remainder there was "a cystic loculation of fluid" without specification as to whether there were one or more cavities. In most cases the contained fluid was under pressure and escaped in a sudden gush on puncturing the cyst wall, leaving a depression on the surface of the cord as if a tumour had been removed, and in some cases permitting the return of a previously displaced cord to its normal position.

The spinal cord itself was frequently described as having an abnormal appearance. The most frequent change was a decrease in the normal vascularity of the pia, and in some instances the breaking down of the arachnoidal adhesions led to an immediate return of the normal pial circulation. In some cases, on the other hand, engorgement of the pial veins was seen. Shrinkage in size of the spinal cord was commented on in six cases.

The posterior spinal roots showed abnormal appearances in six cases, the most striking being a matting together of the roots by arachnoidal adhesions; this was particularly noticeable where the cauda equina was involved. In some cases the nerve-roots appeared shrunken, and in others they were swollen, hyperæmic, and beaded in appearance.

To summarize these observations: Operation commonly showed a dura mater which pulsated defectively and a thickened arachnoid bound by adhesions to the overlying dura and more often still to the underlying pia. These arachnoidal adhesions were in some instances associated with solitary or multiple cysts, which in their turn compressed the cord. Further, the adhesions often disturbed the normal vascular arrangement of the cord; then the latter presented a pale, shrunken appearance.

The following operation notes illustrate the changes seen on laminectomy:—

Case 10.—"The arachnoid from end to end of the incision was uniformly thick and opaque, looking like a wet blanket enveloping the cord. On incising it, clear cerebrospinal fluid escaped. The cord appeared pale and shrunken, and yet the arachnoid was only slightly adherent to it. The thickening of the arachnoid involved the covering of the roots and of the ligamentum denticulatum."

Case 11.—"Adherence of the arachnoid to the dura prevented the exposure of the former without escape of cerebrospinal fluid. After opening the dura the cord was seen to be pushed backwards by something lying anterolaterally and presenting between the 6th and 7th dorsal roots on the right side. On probing this there was a sudden gush of fluid from what appeared to be a tense cyst. The cord immediately sank back, assuming a normal appearance and pulsating naturally."

Case 15.—"The arachnoid appeared normal, but was very tightly distended with cerebrospinal fluid. There was no evidence of loculation, but at the level of the 6th dorsal segment there was an incomplete septum between the arachnoid and the posterior surface of the cord. The cord was pale and shrunken and the pia remarkably devoid of blood-vessels."

Case 19.—"The membranes were all matted and there was no general subarachnoid space. The nerves of the cauda equina were matted into a solid bundle. Opposite the arch of the 10th dorsal vertebra on the left side there was a cyst with a fairly thick wall, fusiform in shape and some $1\frac{1}{2}$ in. long. It lay on the left of the cord which was compressed exactly as by a tumour. After emptying the cyst the cord was 'cupped' and it expanded considerably during the rest of the operation."

Case 35.—"On opening the dura an opaque band of arachnoid was found constricting the cord at the level of the 4th cervical vertebra and extending up and down for $\frac{3}{4}$ in. When it was severed the cord, which was previously constricted in that region, regained its normal vascularity."

The literature contains but few cases which were submitted to a careful autopsy, or in which tissue removed at operation was examined microscopically.

Grinker [19] describes a case of meningitis serosa circumscripta in which the lumbo-sacral enlargement and cauda equina were involved, and which died after operation. At autopsy the spinal leptomeninges appeared slightly thickened and opaque. The cord appeared normal except in the conus medullaris which was soft. The cauda equina was normal except for dilatation of its veins. Microscopically the cord showed no evidence of tumour, cavitation or collections of unusual cells. Weigert preparations and sections stained for fat were normal, but Nissl preparations showed some chromatolysis in the cells of the lumbo-sacral enlargement. The pia mater was normal, but the arachnoid and perineurium were severely altered. They were thickened by increase of connective tissue, the blood-vessels were dilated and the tissue contained excess of pigment. There was no perivascular infiltration with leucocytes.

Kron [21] describes a case in which some of the small opaque areas

found in the arachnoid at operation were excised for microscopy. They consisted of piled-up layers of connective tissue in interlacing bundles of unequal size. In places there was a rich round-celled infiltration.

Marinesco, Draganesco and Hornet [28] describe the post-mortem findings in a case in which the lumbo-sacral enlargement and cauda equina were involved in a patient giving a past history of syphilis. The patient died eighteen months after operation. Microscopically the dura appeared normal. The pia-arachnoid was a little thickened in the mid-dorsal region and greatly so in the region of the lumbo-sacral enlargement and cauda equina. It was adherent to the dura and contained a number of small milky plaques. The cord itself was atrophic in its lower part. Microscopically the dura was normal but the pia-arachnoid was greatly thickened. The fila of the cauda equina appeared as if embedded in celloidin. The newly formed tissue consisted of fibroblasts infiltrated with lymphocytes and contained proliferating blood-vessels. There was thickening of the adventitia of the vessels and hyaline degeneration of their walls. The cauda equina showed some increase in the number of nuclei of the sheath of Schwann. The cord itself had suffered some peripheral demyelinization in the lumbo-sacral region, and an area of softening with degeneration of the ganglion cells was present at the 10th dorsal segment.

My own material for microscopical study is unfortunately scanty and adds little, if at all, to our understanding of the cause of the disease.

It consists of material removed at operation in two cases (35 and 41) and full microscopical examination of the cord removed at autopsy in one case (32).

Case 35.—Portions of the opaque bands of arachnoid, found at operation to be constricting the cord, were removed. Microscopically they were found to consist of hyaline connective tissue with an inner layer of endothelial cells. The connective tissue was very much thicker and less cellular than the normal arachnoid, and in one or two places its fibres were formed into rounded knots of hyaline material. There was no cellular infiltration.

Case 41.—Portions of the spinal muscles, extramedullary fat, dura mater and arachnoid were removed for examination. The muscle fibres were normal except that chains of nuclei were seen at the insertion of several fibres. One or two areas of dense lymphocytic infiltration were present in the perivascular lymphatics. The extramedullary fat, dura, and arachnoid appeared normal.

Case 32 (*Summary*).—Male, aged 52. Nine months' history of increasing weakness of legs and paræsthesiæ of arms. Signs of spinal compression at C.4 segment. Cerebrospinal fluid was at first normal, later showed

increase of protein. Laminectomy revealing arachnoidal cyst. Death. Autopsy.

H. B., a male boot operative, aged 52, was admitted to the National Hospital, Queen Square, under the care of Dr. Holmes, on April 4, 1929. He complained of weakness of the legs of nine months' duration. He had acquired gonorrhœa in his youth and twenty years before had suffered an attack of sciatica on the right side which had cleared up after nine months and had not recurred. Three years before admission he had several carbuncles. In other respects his past health has been good and his family history was uneventful.

His illness began nine months before admission with involuntary spasms of the legs at night. Three months later the legs became numb and tingling after walking, and his gait became unsteady, especially in the dark. At this time a purulent discharge occurred from the right ear, followed by deafness on that side. Two months before admission his legs began to tremble, and one month later a numb sensation began in the fingers of both hands and spread up the arms. Slight delay in starting micturition was noticed, but there were no other symptoms.

On examination the functions of the cranial nerves were normal except for deafness in the right ear from chronic otitis media and slight nystagmoid jerks on full lateral deviation of the eyes to right and left.

There was slight general atrophy and weakness of the muscles of both arms but especially of the triceps. Fibrillation was seen in the deltoid and triceps of the left side. The musculature of the trunk was normal, that of both lower limbs showed slight general weakness.

The tendon reflexes were symmetrically accentuated throughout the body, the abdominal reflexes were diminished on the right and absent on the left, and both plantar responses were extensor.

Sensibility to pin-prick was diminished along the ulnar borders of both hands, forearms and arms and upon the trunk from D.2 to D.8 segments and D.10 to D.12 segments, and on the legs below the middle of the thighs. Sensibility to light touch and temperature was everywhere normal, and the appreciation of passive movement and vibration and compass point discrimination were undisturbed. His gait was ataxic.

The cerebrospinal fluid contained 0·05 per cent. protein and 2 cells per c.mm. Qualitative tests for globulin were negative, as was the Wassermann reaction. Lange's colloidal gold curve was 0001210000. The pressure of the fluid was not estimated. The blood Wassermann reaction was negative. The blood-count was normal. An X-ray of the spine showed some osteo-arthritis. Under observation the wasting and weakness of the upper extremities increased, while the legs became more spastic. The areas of hypalgesia extended until there was a general diminution below C.7 segment on the right and C.8 on the left. Lipiodol was introduced into the cisterna magna and descended normally.

The patient was discharged to be kept under observation, but was readmitted on May 27. The symptoms had steadily increased. The

paræsthesiæ in hands and arms were more severe and the legs weaker. Considerable pain was experienced in the lower abdomen and the thighs.

The wasting of the hands and forearms was more evident and the sensory loss to pin-prick had extended up to the C.3 segment and there was diminution of sense of temperature and light touch below C.4 segment. The cerebro-spinal fluid was again examined. The initial pressure was 200 mm. Queckenstedt's test negative. Total protein 0·85 per cent. Cells 4 per c.mm. Globulin was present in excess. The Wassermann reaction was negative in blood and fluid.

The diagnosis of intramedullary tumour was considered most likely, but syringomyelia and meningitis serosa circumscripta were considered as alternatives. It was decided to explore the spinal cord with C.3 and C.4 segments as the centre of the operative field.

On June 21, 1929 laminectomy was performed, the laminæ of the cervical vertebræ 1 to 5 inclusive being removed. The dura was tense and filled with spinal canal but it pulsated normally. It was opened without injuring the arachnoid which was quite translucent—without any opacities.

The cord was seen to be pushed backwards and a probe was passed in front of the cord without injuring the visible portion of the arachnoid. Immediately there was a gush of clear fluid and the cord resumed its normal position. The arachnoid still surrounded the cord as a translucent distended membrane and no blood entered its spaces. The condition was considered to be one of meningitis serosa circumscripta and the wound was closed in the usual way.

The patient died two days later from bronchopneumonia and a partial post-mortem examination was made as removal of the brain and spinal cord only was allowed. The brain appeared to be normal in every way. The spinal cord was distorted in shape at the level of the 4th cervical segment in such a way as to suggest that it had been compressed from its right lateral aspect. Below this level the cord was normal in appearance and no alteration in the pia or arachnoid could be detected in any part. The cord was fixed in 10 per cent. formol saline and sections of each spinal segment prepared and stained by Weigert's method and with haematoxylin and van Gieson's stain.

Weigert preparations.—In segments C.1, C.2, C.3 there was marked distortion of the normal shape of the cord which was flattened from side to side. There was a marginal zone of pallor in each lateral column and the bundles of myelinated fibre were broken up as if from trauma.

The most striking change, however, was seen in the posterior columns. The columns of Goll were well preserved but on both sides there was a band of pallor in the columns of Burdach in their medial part, which began near the periphery and extended in increasing intensity towards the posterior grey commissure. In the more anterior part the areas of degeneration on either side converged towards the mid-line until they reached the postero-median septum and the posterior grey commissure.

The descending tracts in these segments stained normally. In the 4th cervical segment the changes were more marked. The band of degeneration

in the columns of Burdach had become more intense and there was pallor of one column of Goll. The entering posterior roots showed demyelinization but the descending tracts were preserved.

At C.5 the cord had assumed its normal contour. The areas of degeneration in the columns of Burdach persisted and the entering posterior roots showed marked pallor. The columns of Goll were normal and apart from a marginal zone of pallor the anterior and lateral columns were unchanged.

At C.6 the picture had changed. The areas of degeneration in Burdach's columns had almost disappeared except near the posterior grey commissure. The entering roots appeared normal. There was now marked pallor in the lateral columns especially in the zone occupied by the pyramidal tract and to a less degree throughout the antero-lateral white columns. The appearance of C.7 and C.8 were similar and in D.1 segment the pallor of the lateral columns and on either side of the anterior median fissure was very marked. In this section, however, the posterior roots again showed degeneration and there was a circumscribed area of pallor, almond shaped, in each column of Burdach.

These areas grew less evident in the sections of segments D.2 and D.3 and from D.4 to D.7 the picture was that of an uncomplicated descending degeneration. The posterior columns stained normally, the pyramidal tracts showed marked pallor and the antero-lateral columns a slight degree of demyelinization.

These changes persisted throughout the lower segments of the cord, but at D.8 a small area of pallor was to be seen in one column of Goll in the immediate vicinity of the posterior median septum. This area increased in size as the cord was descended until D.11 segment but afterwards decreased until the S.3 segment where it disappeared.

The appearance of the sections stained by Weigert's method suggest that the cord had been subjected to compression which was most intense at the 4th cervical segment. Above this level there was degeneration of the ascending tracts, below it of the descending tracts. In addition over several segments of the cervical cord the incoming posterior roots showed degenerative changes.

Sections of each spinal segment were stained with hæmatoxylin and counterstained by van Gieson's method. There was no abnormal thickening of the pia at any level and the blood-vessels throughout appeared normal. There was no cellular infiltration of the leptomeninges and no increase of cells in the perivascular spaces of the cord.

The substance of the cord itself appeared normal at all levels except in the upper cervical region at which there was some marginal rarefaction of the white matter where fibres had disappeared with very little glial proliferation. The nerve-cells of the cervical and lumbar enlargements were normal.

The pathological findings in this case are such as might be expected in any case of spinal compression above the cervical enlargement and

cannot be said to add any positive facts to our knowledge of the nature of meningitis circumscripta. Taken in conjunction with the clinical and operation findings they do however show that severe spinal compression may be caused by an extremely local change in the pia-arachnoid without any change being detectable in the remainder of the cord or its membranes.

Consideration of the operative findings and of the pathological material available from the present cases and from the descriptions in the literature, leaves little doubt that the essential change in meningitis serosa circumscripta is a morbid process in the spinal leptomeninges, whereby abnormal adhesions and bands are formed in the subarachnoid space which may or may not be associated with cystic accumulation of fluid in their meshes.

These adhesions owe their existence to a low-grade inflammatory process which is probably of diverse origin. They vary in degree from single bands and diffuse fine strands of fibrous tissue to dense adhesions obliterating the subarachnoid space and enveloping the emerging nerve roots. From whatever cause the meningeal adhesions arise they produce their symptoms by disturbing the functions of the spinal cord or its nerve-roots by direct mechanical pressure and by interference with the vascular supply of the cord which leads in its turn to intramedullary changes.

Stookey has suggested that the intramedullary changes result from the repeated minor traumata which are inflicted upon an anchored cord by the normal spinal movements, but this theory seems to have no advantages over the simpler one of medullary ischæmia, and it fails to explain convincingly those cases where the process remains strictly localized.

Post-operative Course of the Disease and Prognosis.

The prognosis in cases of meningitis circumscripta has been the subject of widely differing estimates by those who have written on the subject. In many isolated cases, particularly where a localized cystic change was found, complete recovery has followed relief of the spinal compression. Horsley considered that the prospect of recovery was good in middle-aged subjects, but poor in the elderly. Stookey considered the prognosis to be favourable if all the adhesions could be severed. Vincent found it to be variable, and stated that when intramedullary changes had occurred little improvement could be expected from operation.

In order to throw light on this aspect of the subject an attempt has been made to trace the subsequent history of the patients referred to in this paper. A record up to the time of death, or until 1935, has been obtained in twenty-four cases. The remaining eighteen cases have been lost sight of at varying intervals after their operations.

Of the twenty-four cases whose subsequent course is completely known five died within three weeks of their operation and may be regarded as direct operative deaths. Eight cases are known to be now dead. Four of them lived less than one year, one survived three years, others nine and ten years respectively. In one case the date of death is unknown. Eleven cases are known to be alive. Of these, four are the same or worse than at the time of operation, and are incapacitated by their disease, but seven have made a complete recovery and are able to work with minor disabilities in some cases. The periods which have elapsed since their operations are fifteen, twelve, eight, eight, seven, five and four years respectively, so that it seems reasonable to regard their relief as a cure. Thus in only seven out of these twenty-four cases (29·2 per cent.) could the outcome be regarded as satisfactory.

It is much more difficult to assess the outcome of the seventeen cases of which trace has been lost. In five of them no information is available. Three, when last heard of, were the same or worse than at the time of operation. Nine cases when last heard of were better than at the time of operation, and in five of these the observation was made two or more years after the operation and may be regarded as satisfactory evidence of recovery. In four cases the period of observation was too short to permit of reasonable judgment.

Thus in the seventeen cases ultimately lost sight of there is reason to think that the outcome was favourable in five (29·4 per cent.), which agrees closely with the more accurate figures available in those cases where full records are available.

The twelve cases (seven certain and five probable) in which the outcome was favourable have been examined to see if there is any common feature which may indicate a good prognosis. Possible underlying causes did not appear to influence the prognosis. In two cases there was a history of syphilis, in two of some other infection, while in six no causal factor could be ascertained.

The duration of the illness before operation was of more significance. In eight of the twelve favourable cases symptoms had been present for less than one year, while in only one had they been present for more than three years. The average duration of these cases before operation was

11·5 months, compared with 24·9 months of the whole group, suggesting that a short pre-operative history is a favourable feature in the prognosis.

The changes found at operation are also an important indication of the probable outcome. In all twelve cases diffuse or localized arachnoidal adhesions were present, in four there was definite cyst formation, and in three a single arachnoid band was found constricting the cord. In none of them was there described any abnormality in the appearance of the cord itself (e.g. pallor or shrinkage), and in several it was specifically stated that the cord appeared normal. It seems, therefore, that the cases which do well share in common the fact that there were no visible changes in the spinal cord itself. On the other hand five of the six cases in which the cord was shrunken had an unfavourable outcome, and none of the cases in which pallor of the cord or avascularity of the pia were noted at operation appear in the group of cases which did well.

Obvious changes in the spinal cord at operation thus indicate a grave prognosis.

Summary.

(1) The clinical and operative findings of forty-one cases of meningitis serosa circumscripta are analysed.

(2) While the pathogenesis is often obscure it is suggested that trauma, meningeal infections, and syphilis are among the causes.

(3) The essential change is the development of adhesions and cysts in the spinal subarachnoid space which produce secondary intramedullary changes by compression and ischæmia.

(4) The symptoms and signs closely resemble those of spinal compression by tumour, but certain points of value in the differential diagnosis are noted.

(5) The prognosis must be regarded as grave, but is least so in those cases which are operated on early before irreversible changes have occurred in the cord itself.

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SPINAL COMPRESSION CAUSED BY ECCHONDROSIS OF THE INTERVERTEBRAL FIBROCARTILAGE: WITH A REVIEW OF THE RECENT LITERATURE.

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INTRODUCTION.

DURING the last few years a fair number of cases of spinal compression due to cartilaginous outgrowths of the vertebral discs have been reported. While working at the National Hospital I had the opportunity of studying three such cases, and by the kind permission of the medical staff I have been able to search the records of the Hospital and to present here ten unreported cases. Fifty odd cases have been also selected from the literature, and on this material an attempt has been made to furnish a systematic analysis of the pathology and the clinical features of the condition.

At odd times cases of spinal compression due to large cartilaginous growths arising from the vertebræ or invading the spinal canal from neighbouring structures have been reported. These growths, which were usually multiple and associated with similar easily palpable subcutaneous growths, are not to be confused with those discussed in the present article, which are small tumours arising from the intervertebral discs, usually slightly lateral to the midline, remaining behind the median longitudinal ligament and the dura, varying in consistency and composed of cartilage, fibrocartilage or tissue analogous to the nucleus pulposus, and under certain circumstances becoming calcified or ossified. These have been described under various names as enchondroma, cartilage nodes, fibrocartilaginous extensions of the disc, ecchondrosis and herniation of the nucleus pulposus.

One of the first references to any type of chondroma compressing the cord is in Oppenheim's textbook [1], where he states that Sich, in 1904, removed a spinal chondroma. About 1911 Middleton and Teacher [2], in Scotland, and Goldthwaite [3], in America, recorded cases of spinal compression due to changes in the intervertebral discs. It is interesting how many years passed before similar cases were reported. Potel and Veaudeau [4], in their review of spinal tumours in 1913, mentioned

only two, Delaginière [5] in his thesis of 1923 reported but two, and Elsberg [6], in 1925, one only.

It is probable that to Stookey [7] the most credit for the presentation of this condition as a clinico-pathological unit should be given. From the time of publication of his article in 1928 many cases have been reported and a fairly large amount of literature written. Other pioneers in this field were Schmorl and Andrae, who in 1929 published the results of routine post-mortem examinations and brought forth the idea of herniation of the nucleus pulposus as the underlying cause. Since then numerous writers have supported this theory, until at the present time it is definitely the accepted one.

Anatomy.—The vertebral bodies, as described in "Gray's Anatomy," articulate with each other in a simple fashion; between them is a pad, the intervertebral disc, and they are held together by that structure and two longitudinal ligaments, the anterior and the posterior. In the cervical region the latter ligament is broad and of uniform width; in the thoracic and lumbar region it is denticulated, being broadest over the disc and narrowest over the vertebral bodies.

The intervertebral discs form the chief bond of union, adhering firmly to the thin layer of hyaline cartilage which covers the adjacent surface of the vertebræ and uniting with the longitudinal ligaments. Each is composed of a peripheral area of fibrocartilage and fibrous tissue called the annulus fibrosus. In the central portion, and posterior to it, lies a soft, pulpy, highly elastic tissue which is expansile and bulges on section; this is called the nucleus pulposus.

The cartilage lining the vertebræ is hyaline with a clear matrix, containing little groups of two to eight cells surrounded by a deeply staining capsule [11]. The annulus is composed of fibrocartilage; it looks much like fibrous tissue embedded in a uniform matrix, but the cells are true cartilage cells, not fibroblasts.

The nucleus pulposus, according to various authors [12, 13, 14, 15], acts as a buffer and a shock-absorber to the spine; it is composed of 75 to 90 per cent. of water, being a semifluid substance containing loose, fibrous tissue and fibroblasts, normal and degenerating cartilage cells, and peculiar large cells derived from the notochord—the "phylasides of Virchow." The latter are masses of cells with large vacuoles containing glycogen in their cytoplasm and often as many as six to eight nuclei.

Frequency.—The incidence of any lesion is not proportional to the number of cases reported in the literature. More interest and a greater

refinement of technique will bring forth many cases which would have been missed under earlier conditions. Indeed, very few are noted in the earlier literature—in a series of 300 verified spinal cord tumours in 1913 [4] there were 107 tumours arising from the vertebræ and only 2 per cent. of these were benign chondromata. In another series of 330 cases collected in Philadelphia, Steinke [17], in 1918, found 14 per cent. were primary vertebral tumours, and of these 50 per cent. were sarcomata. However, in his review he presents six cases of endochondroma, approximately 2 per cent., but he gives no details regarding them. In the next series of note, that by Elsberg in 1925 [6], we find that 20 per cent. were extradural tumours with only one chondroma. However, in his next publication, during 1931, he recorded 40 per cent. of extradural tumours, and 14 per cent. of these chondromata. During the last few years several articles have appeared dealing with this condition, the most important being those by Stookey [7] reporting seven cases, Elsberg [18] reporting fourteen, and Mixter and Barr [12] nineteen. In all, fifty to sixty cases are reported in different articles; as far as their frequency can be estimated the figure of 14 per cent. of all spinal cord tumours is probably too high, it probably lies between 5 and 10 per cent.

Little was known of the intervertebral disc until Schmorl [8] routinely removed the spine at autopsy. In 2,000 cases he discovered 737 cases in which the disc cartilage herniated either into the structure of the vertebræ or posteriorly under the longitudinal ligament. This work was confirmed to a great degree by Andrae [9], who found in 368 autopsies posteriorly-directed extensions of the disc into the spinal canal, in 15 per cent., though very few of these were large enough to cause symptoms. It must be realized that cartilaginous extensions are fairly common, and that many may produce no symptoms and may only be found at routine autopsy.

Ætiology.—It is perhaps under this aspect that the most recent and interesting work has been done. There are two main theories: the original believes that these lesions are neoplastic in nature, hence they are given the name "chondroma"; according to the more recent theory they are herniations of the nucleus pulposus through the annulus fibrosus with secondary changes in the herniated tissue. As the differentiation has been done from the microscopical examination of these lesions, it is advisable to review briefly the structure of an ordinary chondroma and of the normal nucleus pulposus.

The chondroma [19, 20, 21] is firm in consistency, bluish grey,

translucent and usually encapsulated. The microscopic appearance is much like normal cartilage, but does not usually have as regular an arrangement. The matrix has often a definitely fibrillar structure, and the whole tissue may resemble hyaline or fibrocartilage. The nuclei are often in little groups of two to eight cells within a fairly definite capsule, giving the impression of a large multinucleated cell. Fixation and staining often cause them to shrink and assume a vacuolated appearance. In differentiation it may be impossible to separate a chondroma from normal fibrocartilage. Chondromata and the normal nucleus pulposus are easily distinguished, but the differentiation between chondromata and the altered calcified nucleus pulposus is difficult. The large groups of neoplastic cells resemble the notochordal remnants, " the phylasides of Virchow," and unless specific stains, such as that for glycogen, are employed it may be impossible to make an accurate diagnosis.

Most of the earlier writers [4, 6, 7] regarded the tumour as neoplastic, and the tissue removed at operation was certainly compatible with the diagnosis of chondroma. One gets the impression, however, that in many instances this diagnosis was made, not because they were certain it was a chondroma, but because they knew of no alternative diagnosis. The chief points in favour of this view were the microscopical appearance and the long history, suggestive of a benign slowly growing neoplasm. The large multinucleated cells confused some authors, and vague mentions of possible chordoma are found in the early papers. Elsberg [18], in one of his later articles, shows some doubts though he classes them as enchondromata, feeling that they are probably neoplastic but admits the possibility that they are merely due to hypertrophy of the disc. As shown, many of his sections are almost identical with areas of normal disc, but in some there is an increase and change in the cells, but this might be matched from selected areas of normal disc tissue. Alpers, Grant and Yaskin [23] are definitely in favour of this theory, as they feel that the variation in size and immaturity of the cells, with the multinucleated cells and the gradual change of the tissue to normal fibroblasts at its attachment of the disc, indicate a neoplastic process. The gradual onset of symptoms suggests a slowly growing neoplasm, for it is hard to believe that a prolapsed nucleus undergoing changes that solidify it but do not increase its size would, over years, produce signs of compression.

The more recent theory, that of herniation of the nucleus pulposus, was originally proposed by Schmorl [8] and Andrae [9]. They showed

that cartilage nodes, single or multiple, are often found in people over the age of 50. These arise under the posterior longitudinal ligament, usually near but not in the midline, and are most frequent in the thoracic and upper lumbar areas, although they have been found in the cervical and lower lumbar regions. They are small, smooth and vary in consistency, and have a definite communication with the nucleus pulposus. Microscopical examination shows a defect in the annulus fibrosus and a prolapse of the nucleus pulposus through this defect. Such a node may resemble the normal nucleus pulposus, or this tissue which has undergone cartilaginous, calcareous or degenerative changes. Peet and Echols [24], Sashin [14], Calvé and Galland [25], Alajounine [26], Petit-Dutailles [28, 29], Mixter and Barr [12], Keyes and Compere [16], and Middleton [2] accept this view.

The mechanism of the production of the node may be considered under the following headings:—

(*a*) The nucleus pulposus is definitely expansile and projects above the cut surface of the disc at autopsy. Keyes and Compere [16] have drilled holes through the annulus fibrosus of animals or cracked it by means of a chisel, and in those which survived for a few weeks necropsy revealed herniation of the nucleus pulposus beneath the longitudinal ligament, producing small nodular swellings.

(*b*) The annulus fibrosus is thinnest and smallest in its posterior portion, and here the disc would be weakest when involved by any diffuse pathological process.

(*c*) The changes in the annulus are produced by trauma in a tissue which has been weakened by degenerative changes. Many of the cases have a history of serious or slight injuries. In support of this is the work of Middleton and Teacher [2] who removed a portion of the spine at autopsy and by means of a press exerted heavy vertical pressure on it. Under this they saw a nodule develop beneath the posterior longitudinal ligament, and later section of the spine showed that the annulus fibrosus had ruptured posteriorly with herniation of the nucleus pulposus.

(*d*) Several facts are in favour of degenerative changes. Alajounine [26] in some of his cases noted thinning of the posterior portion of the annulus with a brownish and degenerated nucleus pulposus. The majority of these cases occur in later life when degenerative changes in other systems are present and recognizable; pure chondromata, on the other hand, are usually found during the adolescent period, and cease growth with the cessation of general growth.

(*e*) Herniation takes place beneath the denticulate posterior longi-

tudinal ligament, and is most pronounced in the weakest areas, namely, lateral to the midline; this explains the usual slightly lateral position of these structures.

(*f*) The structure of the node is that of the normal nucleus pulposus, or of a nucleus pulposus which has undergone degenerative and hyperplastic changes. The structure of pure nucleus pulposus is rare, the usual changes being either the laying down of a dense cartilaginous matrix producing a tissue resembling fibrocartilage, or, in some cases, the occurrence of areas of calcification or ossification producing a firm hard

FIG. 1.

FIG. 2.

FIG. 1.—Section from a portion of the nodule of Case 4, showing a structure very like normal hyaline cartilage.

Fig. 2.—Section from the nodule of Case 5, showing a structure similar to normal fibrocartilage.

node. To support this many authors lay stress upon the microscopical picture, and particularly on the presence of large notochordal cells. As we have seen previously, similar cells may occur in chondromata; hence the microscopical picture cannot be considered as definitely diagnostic.

In 80 per cent. of my own cases the tissue resembled hyaline or fibrocartilage; in the other two it contained more nuclei with a fair number of groups of cartilage cells, but even these were not cellular when compared to a typical chondroma arising elsewhere in the body. In no case was any tissue resembling the nucleus pulposus or cells of

Virchow found. In general, the structure suggested an origin from the nucleus pulposus with later hyperplasia and fibrocartilaginous changes.

Pathology.—To the naked eye the tumour lies extradurally and beneath the posterior longitudinal ligament, usually slightly lateral to the midline. It is small, 1 to 2 cm. in size, smooth, rounded, often slightly lobulated or oval, and whitish or yellowish white in colour. In consistency it varies from extreme softness to a stony hardness. It is usually encapsulated and in many cases there is a communication between the nucleus pulposus and the tumour.

The microscopical section is varied as the tumour may consist of hyaline cartilage (fig. 1), fibrocartilage (fig. 2), calcified or ossified cartilage, tissue resembling the nucleus pulposus, or tissue consisting of dense fibrous stroma.

Cases.

These cases are all taken from the records of the National Hospital, Queen Square, or were personally observed there in the wards. In all cases but one pathological verification has been obtained.

Case 1.—L. H., female, single, aged 46, was admitted on February 28, 1918, under the care of Dr. James Taylor.

There was no history of any previous injury. In the summer of 1914 she developed sharp shooting pains in the stomach. These soon went away after an abdominal operation, and she remained free from pain from August, 1914, to December, 1916. At this time similar pains, increased by movement of the back, occurred in the right side, and her appendix was then removed. Following the operation the pain slowly disappeared, but returned in June, 1917 and was attributed to post-operative abdominal adhesions. This pain persisted and later became localized in the lumbar region, with a definite radiation to the front of the abdomen. In September, 1917, her legs became weak and stiff, and about this time she fell, fracturing the right leg. During the last month before admission she complained of coldness of her legs and defective control of the sphincters.

General examination revealed only some diffuse non-localized spinal tenderness. The neurological examination revealed normal cranial nerves. There was a slight elevation of the umbilicus when she raised her head and there was a spastic paralysis of her legs, which assumed a position of flexion and adduction and usually showed constant reflex flexor spasms. The sensation to light touch was impaired below the umbilicus and was absent below the first lumbar area. Sensation to pin-prick and to heat and cold was impaired below the umbilicus, and absent below the knees. There was no appreciation of either vibration, position, or deep pressure in the legs. The lower abdominal reflexes were absent, whilst the upper ones were present. The knee and ankle jerks were exaggerated, and the plantar responses were both extensor. There are no available reports of radiography or analysis of the cerebrospinal fluid.

On April 4, 1918, the theca was opened opposite the bodies of thoracic 7 and 8, and a large fusiform cyst of the cord protruded. This burst, discharging a clear fluid. Following this the cord was pulled aside and a hard tumour, the size of a pea, was removed from these vertebræ. This was reported to be cartilaginous tissue with areas of calcification.

Following the operation there was no improvement, and her general condition on discharge was worse than on admission.

Discussion.—There is no doubt that the symptoms were caused by the cyst. However, the earlier symptoms were undoubtedly produced by the cartilaginous node and possibly continuous injury to the cord might have resulted in the cyst. There may be some doubt as to the propriety of including this case amongst the cartilage nodes, but the node had undoubtedly a great deal to do with the course of the illness, and was not merely an incidental finding.

Case 2.—P. W., male, single, aged 25, was admitted on August 9, 1927, under the care of Dr. C. P. Symonds. He had had congenital cataracts. The right eye was removed at the age of 12. The left eye was operated on at 13 years, and since the last operation he had been almost completely blind. There was no history of trauma.

In December, 1926, he complained of pain in the small of his back and in the right hip which increased on bending, and also of slight numbness of the right knee. In January his legs became weak, the numbness increased, and sexual impotence developed. In June the numbness spread to both buttocks and the genitalia. In July the pain decreased as the numbness spread, and he developed loss of sphincter control.

General examination showed an absent right eye; the left eye was blind with corneal opacities and cataracts.

Neurological examination showed normal cranial nerves. Both legs were weak, especially the left which was also wasted. Sensation to light touch was lost over the saddle area and the genitalia. There was hypalgesia over this area, and at the upper level of this there was a burning reaction to pin-prick. The appreciation of vibration was diminished over the left malleolus, but the sense of position was normal throughout. The knee jerks and ankle jerks were absent and the plantar responses were flexor.

Lumbar puncture showed a low pressure and a cerebrospinal fluid which was yellow, coagulated spontaneously and contained 5 mononuclear cells per cubic centimetre. The total protein was 1·5 per cent., the Pandy 2+, and the Wassermann was negative.

Radiography revealed no changes in the spine.

An operation was performed by Mr. Armour on September 8, 1927, when a large outgrowth of the disc was found protruding between the second and third lumbar vertebræ, displacing the cauda equina backwards. As much as possible was removed by chiselling.

The tissue removed was fibrocartilage undergoing calcification. It was not cellular, but contained a few groups of six to eight cartilage cells in different areas.

Following the operation he lost all power in his legs and sphincters, and the hypalgesia increased. These changes, however, began to show improvement on discharge. He improved, and finally in 1928 could stand. However, he injured his back in 1929, and all his symptoms returned. When he was readmitted to the hospital in March, 1930, his condition was much as on the previous admission with the exception of an increased sensory loss. He was again operated upon and some neuro-fibrocartilaginous tissue was removed, but no chondroma found at this time. There was no appreciable improvement on discharge, but since then some improvement has occurred.

Discussion.—This seemed to be a straightforward case until the second operation revealed the presence of a neurofibroma. However, the earlier symptoms were undoubtedly due to the ecchondrosis, which was fairly large.

Case 3.—J. H., male, married, aged 37, was admitted on November 19, 1928, under the care of Dr. G. Riddoch.

In 1926 he fractured his left leg, and following that he complained of some stiffness of the leg. Six months before admission he began to drag his left leg and found that he could not put his heel to the ground because of pain. This pain increased, particularly in the sacral region, and there were also occasional twinges in the left side of his chest. For several months he noticed numbness of both legs, chiefly of the right, and finally, three months before admission, he developed priapismus and a definite difficulty in micturition.

General examination showed some limitation of movement of the mid-thoracic portion of the spine.

Neurological examination.—His legs were spastic, paralysed, and showed numerous flexor spasms. There was weakness of the lower abdominal muscles and of the intercostals below the 8th dorsal. The sensation to cotton-wool was lost below the 7th dorsal area and impaired for two segments above that level. Pin-prick and the sensation to heat and cold were similarly affected, but there appeared to be less change on the right side. There was no appreciation of either vibration or position below the 5th dorsal area. The lower abdominal reflexes were absent, the upper present. The knee-jerks and ankle-jerks were exaggerated and the plantar responses were both extensor. Frequent reflex spasms of the legs occurred, and priapismus was present at times.

Lumbar puncture showed no pressure. The cerebrospinal fluid was yellow and had 8 mononuclear cells per cubic centimetre. The total protein was 0·8 per cent., the Pandy was 3+, and the Wassermann negative. Radiography revealed a small opacity in the vertebral canal opposite the body of the 7th dorsal vertebra.

An operation performed by Mr. Armour on December 4, 1928, revealed a large mass of cartilage arising between the bodies of the 7th and 8th dorsal

vertebræ. It compressed the cord from behind and from the left, and was removed.

It consisted of fibrocartilage and included areas which had undergone calcification. After the operation he developed meningitis and died. A post-mortem examination of the cord showed the maximum damage to be about the 9th dorsal segment, but a larger area extending up to the 6th dorsal was definitely necrotic.

Discussion.—The history suggested that the tumour dated back six months only, but its size and calcification indicate it had undoubtedly existed for several years, and possibly might be related to the trauma several years before. The loss of sensation several segments above the node has been observed in several other cases, and might be explained by circulatory damage at a higher level than the node.

Case 4.—H. J., male, married, aged 51, was admitted on August 3, 1930, under the care of Dr. M. Critchley.

In 1926 the patient noted a pain in his lower back described as "lumbago," from which he recovered in three weeks. In March, 1930, he had a severe pain radiating from his hips to his toes. His legs were weak and he could hardly walk. Later he found that the pain was increased by coughing and movements. The weakness increased and he was forced to go to bed. Finally his condition became so bad that he could barely move his legs, particularly the left, in bed. About two months prior to admission he noted numbness of his feet, which gradually spread to the thighs and was associated with the disappearance of the pain. One month before admission he lost control of his bowels and his bladder, finally becoming impotent.

General examination showed no intercurrent disease.

Neurological examination revealed normal cranial nerves. Both legs were very weak, more definitely so the left. The tone was less than normal, and both legs were wasted, the left more so. Fibrillation was not present. Sensation to light touch, pin-prick, heat and cold were lost over both saddle areas, the back of the right thigh, both calves and the outer aspects of each leg. The loss was greater on the right side. The appreciation of both vibration and position in both legs was impaired. The right knee-jerk was normal, the left knee-jerk absent, and both ankle-jerks were exaggerated. A bilateral ankle clonus was present and both plantar responses extensor.

Lumbar puncture revealed a complete spinal block with a clear fluid containing 6 mononuclear cells per cubic centimetre. The total protein was 0·2 per cent., with a Pandy 3+ and the Wassermann reaction negative. Radiography revealed osteoarthritis of the lumbar spine.

An operation by Sir P. Sargent on August 8, 1930, showed a uniform swelling of the conus medullaris.

After the operation the pain was less, but power and sensation were not as good. This condition persisted for several months, at which time he developed pyuria with a marked systemic reaction, finally terminating in his death.

At post mortem no tumour was found in the conus medullaris, but from the disc between 1st and 2nd lumbar a small extradural nodule of firm consistency extended. The cord lay, however, comparatively free. The microscopical examination showed that the nodule was fairly cellular fibro-cartilaginous tissue.

Discussion.—This was a nodule producing changes in the conus medullaris. There is a possibility that the "lumbago" four years previously might have been related to this cartilaginous node.

Case 5.—A. L., male, married, aged 44, was admitted on March 5, 1931, under the care of Dr. G. Holmes.

The patient's past history included no evidence of trauma. Five years before admission he noticed some numbness and tingling of the legs and weakness of both lower extremities. At this time he could barely walk, but after three months rest he improved, and was able to resume his work as a shepherd within six months.

There were no further symptoms until March, 1930, when he noticed tingling and numbness of the legs. This spread to the hips and at the same time he became so weak he could not walk. About two months before admission his legs gradually flexed and began to show spasmodic jerkings. For one month he complained of sphincter troubles.

General examination revealed no intercurrent disease.

Neurological examination revealed normal cranial nerves. There was weakness of the lower abdominal muscles and of both legs. The legs were spastic and assumed a position of flexion. Sensation to light touch, pin-prick, heat and cold was impaired below the umbilicus, very poor below the hips, and absent below the knees. Sensation was better on the left side and there was evidence of bilateral sacral sparing, again more marked on the left. The appreciation of both vibration and position were lost in the legs. The upper abdominal reflexes were present while the lower ones were absent. The knee and ankle jerks were exaggerated and the plantar responses were both extensor and associated with a marked withdrawal phenomenon.

Lumbar puncture showed no block, and there was a clear fluid with one mononuclear cell per cubic centimetre. The total protein was 0·09 per cent., with negative Pandy and Wassermann reactions. Radiography revealed generalized osteoarthritis of the spine.

An operation was performed by Sir P. Sargent on April 24, 1931. A firm tumour was found arising from the disc between 9th and 10th dorsal vertebræ, being anterior and extradural in position. This was removed by a gouge. The tissue was reported as fibrocartilage.

After operation he improved for about four weeks but this did not persist and he was sent to a convalescent home. He was readmitted from there in November, 1931, with power absent below the knees and with sensation and sphincter trouble as on the previous admission. He was sent home in February, 1932, and was not heard of again.

Discussion.—This case showed a definite onset five years ago with a complete remission. Because of the trauma of operation and the long duration of the symptoms the improvement was slight.

Case 6.—V. B., male, married, aged 45, was admitted on September 9, 1933, under the care of Dr. J. Collier.

The patient had injured his left cornea at 10 years of age, had fractured both ankles at 23 years of age, and at 25 was in a hospital for pain in the lower back. This required rest for three months and cleared up, but when he was 42 a similar attack occurred requiring two months in bed.

In the summer of 1932 he felt some tingling over the sole of the left foot followed by a similar sensation in, and some weakness of, the fingers of the left hand. During the month before admission he noted that his left foot was numb and cold, that both legs were weak and that finally his right leg became numb. Later the numbness spread to the right lower chest, and just before admission he complained of difficulty in micturition.

General examination revealed a corneal opacity in the left eye.

Neurological examination revealed in the cranial nerves a left pupil which reacted sluggishly to light and fine lateral nystagmus in either direction. There was some weakness and atrophy of the left hand and weakness of the legs, most marked in the dorsiflexors of the left foot. Sensation to light touch was lost on the right foot, while that to pin-prick and heat and cold was absent on the right side below the 5th cervical area. The appreciation of both vibration and position was decreased on the right side. The only reflex changes were bilateral plantar extensor responses.

Lumbar puncture showed a partial block. Cisternal puncture produced a clear fluid with one mononuclear cell per cubic centimetre. The total protein was 0·02 per cent. with a negative Pandy and a negative Wassermann. A later lumbar puncture revealed no block. Radiography showed advanced spondylitis. A lipiodol injection showed some arrest at the level of the 5th cervical vertebra.

An operation was performed by Mr. Taylor on October 3, 1933. The cord was displaced backwards by an extradural mass on the left side arising from the region of the 4th cervical vertebra. It was removed by chiselling. The fibrocartilage presented only a few nuclei.

No change was noted after operation. He was discharged but returned in April, 1934, with more weakness of the left side and a definite right-sided analgesia. After a few months he was again discharged showing a slight improvement. No operation was done at this time.

Discussion.—This was the first case in our series to be verified by lipiodol. It is hard to connect the location of the pain in the early attacks with a node in the cervical region.

Case 7.—L. P., female, married, aged 45, was admitted on July 2, 1934, under the care of Dr. P. Martin.

Seventeen years ago she had her left arm severely crushed in a train wreck. One month before admission she developed a pain in the small of her back. This increased and within two weeks it spread down the right leg and later down both legs. The pain was made worse by walking and coughing, and gradually became so severe that she could not sleep. Three days prior to admission she noticed numbness over the perineum and the backs of both legs. This was soon followed by a loss of sphincter control. There was no definite weakness of the legs.

General examination revealed the amputation of her left arm and an area of tenderness over the sacral vertebræ.

Neurological examination revealed normal cranial nerves. There was some weakness in the glutei and hamstrings of the right leg. There was complete loss of all forms of sensation over the saddle area and the backs of her legs. Both ankle-jerks were lost and the plantar responses were flexor.

Lumbar puncture revealed no block. The fluid was clear with one mononuclear cell per cubic centimetre. The total protein was 0·075 per cent., the Pandy was weakly positive, and the Wassermann negative. Radiography revealed a normal spine. An injection of lipiodol showed arrest at the lower border of the fourth lumbar vertebra.

An operation by Mr. Taylor on August 14, 1934, showed some bulging of the anterior thecal wall and a smooth yellow mass arising from the disc between 4th and 5th lumbar areas. This was removed by dissection and curettage. This tissue was reported as fibrocartilage with few nuclei.

She had a very good post-operative recovery and on discharge was walking well. The ankle-jerks were returning and there was little evidence of sensory loss.

Discussion.—The short history and easy removal produced good results. There was probably no relation between the trauma seventeen years previous and the node.

Case 8.—A. C., female, married, aged 43, was admitted on June 16, 1935, under Dr. G. Holmes.

Seven years ago she complained of "lumbago," which consisted of pain in the small of her back. This recurred five years ago and at this time the pain spread down the right leg. There was no history of trauma to be obtained.

When the pain ceased five years ago the right foot remained numb and this persisted until admission. There was no further pain until January, 1934, when it returned in the same area as previously. About this time she fell and injured her back. The pain became much worse and numbness developed in the soles of both feet spreading to the knees in September, and to the hips in November. In these latter months she noted weakness of the legs and loss of sphincter control with incontinence.

General examination revealed some tenderness over the sacral vertebræ.

Neurological examination revealed a slight bilateral weakness of the flexors

of the hip and adductors of the thighs. Sensation to light touch was normal, but that to pin-prick and heat and cold was decreased between the 10th thoracic and 1st lumbar areas, definitely impaired over the 2nd and 3rd lumbar areas, and slightly on the lower lumbar areas, but intact in the areas of the sacral roots. The appreciation of both vibration and position was absent in both legs. The knee-jerks and ankle-jerks were exaggerated with ankle clonus and bilateral plantar extensor responses.

Lumbar puncture revealed no block. The fluid was clear with no cells and a total protein of 0·1 per cent. The Pandy was 1+ and the Wassermann negative. Radiography revealed osteoarthritis of the lower lumbar spine.

An operation was performed by Mr. Taylor on March 26, 1935. The dura was pushed forward by an intensely hard osteochondroma arising between 1st and 2nd lumbar vertebræ, probably from the disc. Removal was impossible.

After the operation she showed a marked paralysis of both legs and a fairly complete sensory loss below the first lumbar area and a slight sensory loss below the 10th thoracic. At the time of writing some recovery had occurred in both power and sensation.

Discussion.—This case had a very long history dating back seven years with a definite remission. The trauma of operation produced a damage to the cord with an increase of all symptoms. In spite of the lack of a pathological report, the clinical history and operative findings appear diagnostic enough to include this in our present series.

Case 9.—J. K., male, married, aged 59, was admitted on July 6, 1935, under the care of Dr. S. A. K. Wilson.

There was no history of trauma, but about five years before admission he noticed a severe pain in the right calf. This spread to involve the whole of his back and the outer side of the right leg. He was placed in a cast for several months with good results, although the pain tended to return at different times with exercise. In the early part of 1935 he noticed numbness beginning in the right buttock, which spread first to the right foot and then to the left buttock and left foot, and finally to the genitalia. With the numbness pain tended to disappear. A few months later he began to lose control of his sphincters. The sole motor symptoms were only a slight stiffness and weakness of the right leg.

General examination revealed a definite rigidity of the lumbar spine.

Neurological examination showed that the cranial nerves were normal. There was a definite weakness and atrophy in the right leg, and a slight weakness of the left leg. Sensation to light touch, pin-prick, heat and cold was slightly impaired from the 1st to the 4th lumbar areas, and lost below this level. The appreciation of vibration was lost in the right malleolus, while the sense of position was normal throughout. The ankle-jerks were absent and the anal reflexes very poor. Very little plantar response could be obtained.

Lumbar puncture revealed no block. There was a clear fluid with no cells. The total protein was 0·11 per cent., the Pandy 1+ and the Wassermann

negative. Radiography revealed marked spondylitis, and a lipiodol injection was arrested at the level of 4th lumbar vertebra.

An operation was performed by Mr. Jackson on April 9, 1935. The cauda equina was displaced backwards by an extradural mass arising from the region of 4th lumbar vertebra. This was enucleated. The tissue was fibrocartilage with few nuclei and no evidence of calcification.

He improved after the operation and was discharged with a fair return of sensation and good sphincter control, but no change in power.

Discussion.—A typical cauda equina syndrome with a definite five year history.

Case 10.—K. S., female, married, aged 59, was admitted on March 21, 1935, under Dr. G. Holmes.

There was no history of trauma. Ten years prior to admission she noticed that her legs would give out while walking. This increased and the right leg began to drag. One year later she developed severe pain in her right chest, paroxysmal and irregular. This pain shifted and gradually went to the right leg, which at the same time became definitely weaker.

These pains in the right leg persisted with definite exacerbations and became gradually more severe. On admission she was subject to very sharp sudden spasms of pain which were severe enough to make her cry out. There was no definite cause for these and they occurred with a marked periodicity, every two to five minutes, and lasted about one-half to one minute. They tended to remain in the same area, namely, the front of the right leg from the hip to the toes. During the last five years the right leg became much weaker, and during the last six months definite weakness developed in the left leg. Before admission she was bedridden with a marked loss of sphincter control and incontinence.

General examination revealed tenderness of the spine between the 3rd and 5th dorsal vertebræ.

Neurological examination revealed definite weakness of the recti abdominis and the intercostals below the 7th dorsal. There was no power in the right leg and very little in the left. Both legs were in the position of spastic extension. Sensation to light touch was decreased below the 6th dorsal area. There was hyperæsthesia on the 2nd dorsal, slight hypalgesia on the 5th to 7th dorsal areas and almost complete loss below this level. There was some bilateral sacral sparing, more marked on the right side. The appreciation of both vibration and position was very poor in both legs, more so the left. The upper abdominal reflexes were present, the lower ones absent, the knee-jerks and ankle-jerks were exaggerated and the plantar responses were extensor. There was a bilateral ankle clonus and a marked flexor withdrawal response of both legs.

Lumbar puncture revealed a partial block. The fluid was clear with no cells and a total protein of 0·3 per cent. Pandy was 2+ and the Wassermann reaction negative. Radiography revealed an area of calcification in the spinal

canal at the level of the disc between the 6th and 7th dorsal vertebræ and some erosion of the margins of the intervertebral disc.

An operation was performed by Mr. Taylor on April 5, 1935. The cord was displaced by an anteriorly placed extradural mass attached to the disc between the 6th and 7th dorsal vertebræ. It was calcified and lay more on the right side. It was the size of a cherry and was removed by chiselling.

The tissue was reported as normal bone and fibrocartilage with extensive calcification. Its general appearance was like normal disc tissue.

After the operation she had still complete paraplegia with anæsthesia below the 6th dorsal and lost sphincter control. When last seen there was slight improvement in power and sensation.

Discussion.—An unusual feature was the peculiar rhythmical pain which occurred with no apparent cause. It was a very long history, at least ten years, with definite remissions. There was also a peculiar but definite level of hyperæsthesia several segments above the node.

Analysis of Cases.

This analysis is based upon the following fifty-eight cases: Six reported in detail by Elsberg [18], seven by Stookey [7], six by Steinke [17], nineteen by Mixter and Barr [12], two each by Peet and Echols [24], Robineau [30], and Dandy [31], one each by Crouzon, Petit-Dutailles et Christophe [32], Alajounine et Petit-Dutailles [26], Bucy [33], and Alpers, Grant and Yaskin [23]. To these were added the ten cases from the National Hospital, Queen Square.

Age.—The average age was 45 years with a variation from 20 to 63 years.

Sex.—Roughly, two-thirds were males.

Trauma.—Definite evidence of trauma could be obtained in about one-third of the cases, varying from a fractured leg to a severe blow in the back.

Initial symptoms.—These may be divided into the prodromal symptoms which cleared up, and the initial symptoms proper with which the illness began. The prodromal symptoms were described as lumbago, sciatica, or as pains in the back, and as they preceded the symptoms of the spinal disease and corresponded in location to these symptoms, it is probable they were related. Roughly speaking, about one-half of the cases had such symptoms, and the average duration of these was five years though they varied from two to ten years. About 90 per cent. of these prodromal symptoms were sensory complaints, usually pain, the rest some form of paræsthesia. In almost all cases

the initial symptoms proper were sensory, chiefly pain in the back, but some first complained of weakness or stiffness of the legs. The average duration of these symptoms was eighteen months, varying from one month to seven years.

Course of the disease.—The initial symptom, almost always pain, was followed by motor disturbances and in the end by disorders of the sphincters. The interval between the onset of the sensory and the appearance of the motor symptoms averaged about twelve months, and that between the motor disturbances and the lack of sphincter control was ten months. The usual history was an onset with sensory symptoms, then about half way through the illness motor disturbances appeared, and finally only a few months before admission sphincter troubles became apparent. In the rarer cases which began with motor symptoms, sensory disturbances occurred next and sphincter palsies developed last. The earliest sensory symptom was most commonly pain, followed by numbness or rarely by other paræsthesiæ. When numbness ensued the pain usually disappeared, but in a few cases where the numbness was referred to a lower level than the pain both persisted. These pains were often referred to the distribution of the root compressed and as such were a good indication of the level of the lesion. In most cases the pains were increased by movement, coughing, or straining and eased by rest. The paræsthesiæ were generally described as tingling, "pins and needles," or, rarely, by peculiar sensations of cold.

The motor disturbances commonly began as stiffness or weakness of the legs, but in a few the arms were first affected. In almost every case motor symptoms developed with the exception of a few cases where the lesion lay in the lower lumbar region.

Sphincter disturbances were observed in about two-thirds of the cases; it generally commenced as hesitancy of micturition and constipation and proceeded to incontinence and loss of sensation of these acts. In a small number of cases impotence or some sexual disturbance was recorded.

Examination of the patient.—It is an interesting fact that practically no evidence of intercurrent disease was reported. The motor signs depend upon the location of the node. In all the cases except a few in which the cauda equina was involved some evidence of disorder of the motor system could be found. No atrophy of muscles or fibrillation was observed in the series at the National Hospital except in a few cases where the cauda equina was involved. This is surprising as the node lying ventral to the spinal canal must first have compressed the

motor roots. In a few cases, however, particularly those of Stookey [7], definite atrophy and fibrillation were reported.

In addition to the subjective symptoms which have already been discussed objective sensory changes are present in over 90 per cent. of all cases. In those in which the sensation escaped the lesions involved only the cauda equina. In most cases the changes were a definite loss to painful stimuli, to light touch, to the perception of vibration and of the sense of position. In a small number of cases the loss to light touch was much less than the loss to painful stimuli. In very few of the cases was there a sparing of sensation on the sacral areas. In none of the cases at the National Hospital, and in a few only of the total series, were those forms of sensation conducted by the posterior columns of the cord unaffected. This is surprising as from their position it might be expected that these lesions would affect the anterior columns of the cord more severely than the posterior columns. Sensory changes were present at a higher level than the node in several of our cases, in three at least two segmental areas above that corresponding to the lesion. It must be therefore assumed that there were some changes in the cord or roots above the node; these were probably secondary to circulatory disturbances. Three similar cases have been recorded, two by Elsberg [18] and one by Robineau [30], with sensory changes two segments above the level of the nodes.

It is interesting that sensory and motor disturbances were present in every case in which the lesion lay above the lower lumbar region, and that they were absent only in some where the cauda equina alone was involved.

In about half of the cases some tenderness of the vertebræ, or limitation of movement of the spine, could be demonstrated.

Reflexes.—There is nothing specific in the state of the reflexes, they are similar to those obtained in other forms of spinal compression. In cases described by Mixter and Barr [12] in which the cauda equina was compressed the ankle-jerks were absent although there was no definite motor or sensory disturbance.

Cerebrospinal fluid.—In about 25 per cent. of the reported cases there was a complete block, and in about 50 per cent. a partial block. The protein was markedly increased in 40 per cent. of the cases, moderately increased in another 40 per cent. and normal in the rest. In practically all cases the cell count was below 10 per cubic millimetre and the cells were all mononuclears. In no case was the Wassermann reaction positive.

Radiography.—The vertebræ showed no specific changes, but in a few cases osteoarthritis was observed. In seven cases changes could be observed in the intervertebral discs, either calcification of its centre or irregular narrowing of the disc itself. It is probable that such changes could be more frequently seen if attention were specifically directed to them. In two of the National Hospital cases the nodes were calcified and visible, but this has not been reported in any other case. In about 40 per cent. of the cases the fall of lipiodol was arrested at the site of the lesion, and by that technique the level of the node was confirmed.

Location of the nodes, as proven by operation in the National Hospital series, was as follows: 1 case was in the cervical region, 4 were in the thoracic region, and 5 in the lumbar region. When all the cases reported are considered about 17 nodes were in the cervical region, 13 nodes in the thoracic region, 27 in the lumbar region, and 1 in the sacral region. The nodes tend to be more common in certain regions as 10 were found between the 4th and 5th cervical vertebræ, 7 between the 6th and 7th thoracic vertebræ, and 21 between the 3rd and 5th lumbar vertebræ.

Differential Diagnosis.

The symptoms and physical signs are essentially those of spinal compression. The following points are in favour of a cartilage node: a history of trauma preceding the illness; intermittent root pains present for a long period of time, even four to five years, before the onset of spinal symptoms; an initial onset of root pain followed by weakness and later by sphincter troubles. Neither the motor nor the sensory signs are characteristic. The spinal fluid shows no characteristic changes, but radiography may reveal the presence of an altered intervertebral disc or calcification of the node. It is probable that as radiologists pay more attention to the structure of the intervertebral disc earlier diagnosis may be possible.

Prognosis.

An analysis of the cases comprising the whole series throws some light upon the factors which determine the prognosis. On the whole age and sex are of little significance. It might be expected that the prognosis would be more favourable in younger patients, but patients as old as 61 have made complete recovery. The level of the node is more important, as patients with nodes in the cervical region do fairly well after its removal, those in whom the thoracic segments are involved do

very poorly, and the prognosis is most favourable when the node lies in the lumbar region, indeed very few of the latter fail to improve after successful operation. The shorter the duration of the symptoms the better the prognosis. Patients with symptoms other than of pain lasting over one year do not do well, but the occurrence of pain and no other sensory symptoms for years does not preclude considerable improvement after operation. One patient who had pain for five years prior to operation made a good recovery. Weakness and stiffness lasting over a year are signs of a poor outlook.

The consistency of the tumour is important. When the node is calcified or ossified the improvement after the operation has usually been slight, certainly much less than when the tumour removed was soft. The trauma of a severe operation seems to increase the symptoms and may produce an almost complete transverse lesion of the cord. Currettage or dissection produces far better results than chiselling or gouging.

In the National Hospital cases the following figures were obtained: 20 per cent. showed definite improvement, 10 per cent. slight improvement, 20 per cent. no change, and 50 per cent. became worse after the operation. In the total series the prognosis was much better as about 70 per cent. improved, 5 per cent. were unchanged, and only 20 per cent. were worse after the operation. The factors given above explain the discrepancy in the figures.

Conclusions.

(1) Ten cases of spinal compression due to ecchondrosis of the intervertebral disc are reported, and the total of fifty cases collected from the literature have been analysed.

(2) The ætiology of the condition has been discussed; the tumour is probably due to a herniation of the nucleus pulposus through the annulus fibrosis.

(3) The cases have been analysed in the hope of obtaining a characteristic clinical picture, but although this has not been possible a number of suggestive diagnostic features have been indicated.

I wish to express my thanks to the Medical and Surgical Staff of the National Hospital for permission to use the clinical and pathological material on which this communication is based, and to Dr. Greenfield for his help in studying the pathological material.

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PROGRESSIVE DEMENTIA, WITHOUT HEADACHE OR CHANGES IN THE OPTIC DISCS, DUE TO TUMOURS OF THE THIRD VENTRICLE.

BY GEORGE RIDDOCH, M.D.

In the presentation of two cases of tumour of the third ventricle attention will be drawn less to focal symptomatology than to the occurrence of progressive dementia in the absence of severe headache, vomiting, and of either papillœdema or optic atrophy. As will be seen, gross hydrocephalus was present in both cases and was, I believe, the cause of the mental deterioration. A complete dissociation of this kind is not common with intracranial tumours in any situation when the illness has lasted for a considerable time, and all the more with tumours which are so placed as to obstruct the circulation of the cerebrospinal fluid.

Tumours in the third ventricle are characterized, sooner or later, by manifestations of severe hydrocephalus which may be paroxysmal or continuous, depending upon whether the obstruction of the cerebrospinal fluid pathway is intermittent or not. For example, with colloid cysts, especially when anteriorly placed, as in Case 1, paroxysmal attacks of hydrocephalus are common and can sometimes be arrested by altering the position of the head—(Weisenburg [4], Dandy [1], Fulton and Bailey [2]). Since these tumours are benign and slow growing, long intervals may separate the attacks until the blockage of the foramina of Monro, or of the Sylvian aqueduct, becomes complete. On the other hand, when the tumour is posterior and is not pendulous, progressive hydrocephalus develops from either plugging of the aqueduct or compression of it from invasion by the neoplasm of the mid-brain. This result is illustrated by Case 2.

Most of the symptoms and signs of hydrocephalus—severe headaches, vomiting, papillœdema, weakness, unsteadiness, and mental dullness—are usually present in varying intensity and often usher in and remain the prominent features of the illness. If the attacks of hypertension are relatively short, papillœdema may be absent until some permanent obstruction occurs; or instead of swelling of the optic discs, primary optic atrophy with visual field defects, sometimes

bitemporal, may result from pressure on the optic tracts or chiasma by the floor of the distended third ventricle. But the slow development of dementia without headache, vomiting, or alteration in the optic discs or visual fields, as in the cases about to be described, is exceptional.

Case 1.—H. N., male, aged 66. Single. According to his relatives he had always been a dull fellow and had retained his job as a Stockbroker's clerk in his brother's office because of their relationship. His work had never been quite satisfactory. His general health had, however, been good until twelve years ago when having tea in a tea-shop he was seen to fall to the ground and was taken unconscious to Middlesex Hospital. He remained in coma for fourteen hours. Sugar was found in his urine, but it disappeared and, since consciousness spontaneously returned, he was discharged under the diagnosis of an unexplained cerebral attack. On inquiry, his sister said that he had complained of headaches before the attack, but was not known to have had any since then.

He remained well until two or three years ago when he became lethargic, so that he was apt to go to sleep at any time, and he became still more inefficient in his work than he was before. The sleepiness and dullness varied but tended to increase. His brother, who was his employer, found that he was slow, forgetful and inclined to repeat himself. In fact, as was said, he behaved like an old man. He complained of a continual sense of fatigue.

On March 20, 1934, he was admitted to the London Hospital. A week before, he was found unconscious on the pavement with a cut across his left eyebrow and his left eyelids were swollen and blue. He soon partly regained consciousness but since then, until his death on April 11, 1934, he remained dull, sleepy and incontinent. His mental condition varied, but as a rule he could be roused to answer questions and to co-operate in an examination. But his answers, although apparently correct, were slow and he was obviously disinterested in everything that happened to him and around him. He did not make any complaint and denied having headache. His memory was very poor and unreliable for recent and past events and he was quite apathetic. Sometimes he was awake during the day for long periods, but at other times he remained asleep so long as he was left alone. He had an excellent appetite and ate enormously, but had to be fed.

On examination, he was thin and looked older than his age. He had not, however, lost weight. His peripheral arteries were firm and tortuous and his retinal arteries narrow. His heart sounds were feeble and his blood-pressure readings 110/75. His pulse-rate was between 50 and 60 and his temperature subnormal. His urine, blood sugar and blood urea were normal. There was no polyuria or polydipsia. Speech and articulation were normal except that he spoke slowly. It was impossible to chart his visual fields by perimeter, but they were full to confrontation tests. His optic discs were clear and there were no hæmorrhages nor exudates in his retinæ. His ocular movements were full, and diplopia, squint, nystagmus and paralytic ptosis were absent.

His pupils were central and equal but small and irregular in outline, and failed to react to light. The reaction on convergence was poor, perhaps partly from insufficient effort on his part. There was slight left lower facial weakness. The functions of the other cranial nerves were normal.

All voluntary movements of his limbs were feeble and shaky, but there was no dystonia or gross ataxia. He occasionally showed a grasp reflex with his right hand. He was confined to bed and was too feeble to sit up without support.

The tendon-jerks of his upper and lower limbs were present and equal on the two sides but were diminished; his abdominal reflexes were present and equal and his plantar responses were extensor.

Sensory testing was unsatisfactory, but there was no apparent gross loss to light touch, pin-prick, passive movement and postural change.

X-ray examination of his skull was negative.

On admission, he was ordered daily rectal injections of three ounces of magnesium sulphate in six ounces of water and a few days later was lumbar punctured. The cerebrospinal fluid pressure, with the patient in the horizontal position, was found to be 160 mm. of water. (This comparatively low reading in view of the severe hydrocephalus will be discussed later). The fluid contained 0·5 per cent. of protein but did not present any other abnormality.

Although tumour of the third ventricle was suspected, the differential diagnosis from traumatic cerebral lesions in an arteriosclerotic subject was difficult and three weeks after admission Mr. Cairns tapped the occipital horn of both lateral ventricles, and obtained fluid at a depth about 4 cm. from the dura. The pressure was surprisingly subnormal. The fluid contained less than 10 mgm. of protein per 100 c.c. 60 c.c. of air was injected into each ventricle and the ventriculograms showed enormous and symmetrical distension of the lateral ventricles with air in the third ventricle. The foramina of Monro could be seen to be enlarged and the curved anterior end of a tumour in the third ventricle was clearly defined.

Operation for removal of the tumour by opening one lateral ventricle and incising the foramen of Monro was considered but was not attempted on account of the patient's condition. He died two days later.

Throughout the course of his illness his condition varied, but he gradually became worse mentally and physically. Nevertheless, headache, vomiting and alteration in his optic discs remained absent.

An autopsy was carried out on the day after death. There was an enormous distension of the lateral ventricles with thinning of the brain substance and flattening of the convolutions. The third ventricle was also greatly enlarged, especially antero-posteriorly. A round colloid cyst measuring over 2 cm. in diameter was attached to the choroid plexus. It lay behind the foramina of Monro, which were grossly enlarged, and well in front of the aqueduct of Sylvius which was narrowed. It bulged into the lateral ventricles from which it was separated by the roof of the third ventricle. The quadrigeminal plate was flattened and above it were the greatly enlarged posterior parts of the lateral ventricles.

Comments.—In spite of gross hydrocephalus of the lateral and third ventricles there was no thinning of the skull or abnormal convolutional markings. The clinical picture was one of slowly progressive dementia with lethargy of at least twelve years' duration, which varied in severity from time to time but without an acute episode until he had his second attack of unconsciousness. All this indicates that the main explanation of the absence of headache, vomiting and papillœdema lies in the very gradual and intermittent evolution of obstruction to the cerebrospinal fluid circulation. The absence of increased pressure in the ventricles and on lumbar puncture supports this view, although the therapeutic dehydration by hypertonic solutions of magnesium sulphate may have played a part.

The first attack of unconsciousness, which was preceded by headache twelve years ago was presumbly due to acute obstruction, but the cerebrospinal fluid circulation became, after a short time, re-established.

The fact that, according to his relatives, he had always been a dull person, raises the question whether the pathological process dates back longer than the history would at first seem to indicate.

It will be noticed that in this case the abnormal sleepiness is attributed, like the dementia, to general cerebral compression and not to focal disturbance of hypothalamic neural mechanism. I agree with Fulton and Bailey [2] that hypersomnia to have definite localizing value should precede the onset of mental dulness.

Finally, the cause of the hydrocephalus is interesting. Since the third ventricle was undoubtedly greatly enlarged, there must have been obstruction of the aqueduct of Sylvius which could not, however, have been directly due to the tumour when its position is considered. At autopsy, the aqueduct was found to be flattened from above downwards and the quadrigeminal plate compressed due, almost certainly, to the overlying distended posterior parts of the lateral ventricles. This then was the cause of the hydrocephalus of the third ventricle as well as of the pupillary abnormalities, namely smallness, irregularity and reflex iridoplegia.

The mechanism by which the tumour produced hydrocephalus of the lateral ventricles has still to be explained. Its size alone may have impeded the flow of fluid through the third ventricle, but it is likely also that it caused intermittent obstruction of the foramina of Munro at least at a stage in its growth when it was perhaps pendulous, or at any rate more movable than it was at a later stage. If this explanation is valid then the increased size of the foramina has been compensatory.

Case 2.—S. C., male, aged 55. On the whole he had been a healthy man until the onset of the illness about to be described.

Throughout his life he had been remarkably free from headaches, but had been subject to occasional " rheumatic " pains in different parts of his body.

In the early part of the summer, 1933, after a period of hard public work, he complained of feeling tired and of being a little unsteady on his legs. He recognized that his memory was failing and that he was unusually sleepy. In August he went to Wiesbaden, as he had done before, with his wife who wished to undergo treatment. Whilst there, because of slight pain and stiffness in the upper part of the back of his neck, he was persuaded to have a course of baths. This made him still more tired and unsteady and by the time he returned to London in September, 1933, he had become considerably worse. He was more dull, sleepy and feeble. He had lost interest and initiative and his memory was so poor that he continually misplaced things and forgot what he wanted. He was aware of these deficiencies, was worried about them and was afraid that he was going to die.

According to his wife he was different from his usually alert self and was at times confused and had obvious difficulty in thinking.

He became fairly rapidly worse during the two days before I saw him with his doctor on September 30 and had been incontinent. It was found that he had been sweating profusely and continued to do so until he died. In addition there was slight fever for no obvious cause. His weight had not varied much and he had not been abnormally thirsty. He did not think that there was anything wrong with him or that he had been ill. He believed that his memory was excellent and his mind clear, in fact, he seemed to be surprised that he was being kept in bed and that a fuss was being made about him. He denied having headache or any other symptom.

He was in a happy, fatuous mood and laughed in rather a silly way. His memory defect was gross and his power of attention and co-operation poor. He was disorientated but not deluded, except about his own condition. He was a florid, well-nourished man. His heart, arteries and blood-pressure were normal for his age. Articulation was slow and tremulous, but speech was normal. Sensory testing was, because of his mental condition, not very reliable, but to rough tests his visual fields appeared to be full. His optic discs were clear and retinal arteries good. His pupils were moderately dilated, the right being larger than the left, both were irregular and reacted very poorly to light, especially the left, but well on convergence. His ocular movements were full and there was no nystagmus, squint or ptosis. When he spoke there was twitching of his face and his tongue was tremulous, but the functions of his cranial nerves were otherwise normal.

There were general weakness, slowness and gross tremor of his limbs on voluntary movement, but no dystonia or ataxia. The tendon-jerks of his upper and lower limbs and abdominal reflexes were present and equal on the two sides, and both plantar responses were flexor. There did not appear to be gross defect in sensibility to light touch, pin-prick and passive movement and change in position.

A provisional diagnosis of dementia paralytica was made, but the Wassermann reaction was found to be negative in blood and cerebrospinal fluid. On lumbar puncture the cerebrospinal fluid pressure, with the patient horizontal, was over 300 mm. of water and the only abnormalities found in the composition of the fluid were a protein content of 0·075 per cent. and weakly positive globulin reactions.

Four days later slight weakness of the lower part of his face and of his upper limb as well as an extensor plantar response on the right side were found.

On these findings a ventricular tumour was suspected. X-ray examination of the skull showed increased convolutional markings and indistinctness of the posterior clinoid processes. On ventricular puncture the pressure was 110 and the protein content 0·005 per cent. was normal. Ventriculograms demonstrated marked dilatation of the lateral ventricles and the anterior part of the third ventricle.

In retrospect it ought to have been clear that the tumour lay in the third ventricle, but the posterior fossa was explored on October 6 with negative results.

The patient died on the following day.

At autopsy the cerebral convolutions were found to be flattened. Both lateral ventricles were greatly dilated. The third ventricle was also enlarged and in its posterior part there was a tumour which measured 3·5 cm. from before backwards and 2 cm. from above downwards. It extended forward to the level of the foramina of Monro, which were much larger than normal, being 1 cm. by 0·4 cm. Above, the tumour was not attached to the tela choroidea and the anterior pillar of the fornix. Behind it extended into the aqueduct of Sylvius for a distance of 0·6 cm. reaching the level of the junction between the superior and inferior colliculi. Below this part of the tumour was the slightly flattened cerebral peduncles. The antero-inferior border was free. Laterally, the tumour extended into the inner part of each optic thalamus for a short distance. The internal capsules were compressed.

Histologically it was found to be a spongioblastoma multiforme.

Comments.—In this case the clinical picture of progressive dementia, dysarthria, weakness, tremor and reflex iridoplegia without headache or papilloedema, closely resembled that of dementia paralytica running a fairly rapid course, until the blood and cerebrospinal fluid were examined. The key to the correct diagnosis of tumour was the raised cerebrospinal fluid pressure on lumbar puncture.

In the localization of the tumour we were unjustifiably led astray by the evidence of air in the third ventricle in the ventriculograms. Ventriculography has been shown by Dandy to be an aid of great importance in the diagnosis of third ventricle tumours, but to be of any

value the X-ray appearances must be correctly interpreted and the radiographic technique of high standard. Unfortunately, in this case, the films did not show clearly enough that air was present only in the anterior part of the third ventricle.

The emotionalism which was associated with dementia in this case, but not in Case 1, is of interest and was probably due to invasion of the optic thalami by the tumour. Nevertheless, emotional facility when inhibition is reduced, as it is in dementia, is often an exaggerated expression of the natural reaction of the individual. S. C. (Case 2) was by nature a cheerful person, whilst H. N. (Case 1) was dull and apathetic and became more so throughout his illness.

Conclusions.

These cases illustrate the fundamental importance in clinical neurology of the time-factor which determines the presence or absence of many symptoms and bears so much on the evolution of illness.

Contrast the abolition of function below the level of an acute, severe spinal lesion with the slowly developing disturbance of function associated with gradual spinal compression. No better examples could be given of the influence of the time-factor in the development of symptoms than headache and papillœdema in intercranial hypertension. When pressure within the skull is rapidly raised, as from sub-arachnoid arterial hæmorrhage, headache is an almost immediate complaint, before consciousness is lost, and papillœdema, often with hæmorrhages, develops within a short time. On the other hand, slowly increasing pressure from tumour, whether local or general, may for long be unassociated with either of these symptoms. In comparison with the development of focal manifestations, their not infrequent late appearance with meningiomas and slow-growing intracerebral gliomas, especially when anteriorly placed, is familiar to neurologists. But in such cases cerebral compression with impairment of circulation is largely local and at this stage mental deterioration is also absent.

Progressive dementia, without headache and papillœdema, may occasionally result, however, from invasion of both frontal lobes and the anterior part of the corpus callosum by an infiltrating glioma or a centrally placed meningioma, but the dissociation and its duration is again dependent upon the rate of development of the pathological process.

These examples illustrate well enough the point under discussion,

but not in the same striking way as do cases of acute and chronic bilateral internal hydrocephalus in which compression of the cerebral hemisphere is general and symmetrical. When distension of the ventricles is rapid, headache is severe and associated with vomiting and weakness, and there may be loss of consciousness and convulsions. If hydrocephalus is of short duration, papillœdema is commonly absent. Acute transient attacks of this kind may occur with ball-valve tumours which block the foramina of Monro, the aqueduct of Sylvius or the fourth ventricle. When, however, ventricular blockage is very slow and progressive but variable, the process extending over a period of months or years, as in the cases which form the basis of this paper, headache, vomiting and papillœdema may be absent even at a stage when dementia or stupor, general enfeeblement and incontinence are advanced. The severity of these symptoms indicates the degree of reduction of general cerebral function from slow compression.

We are not here primarily concerned with the mechanism by which these disorders of function are produced, but the fact that great improvement may follow relief from compression by removal of its cause is in favour of the view that impairment of blood supply is the primary factor. In the causation of headache, gross variation in tension in the vessels, especially the arteries, and venous sinuses and dural septa, probably play an important part.

Lastly, I have purposely paid little attention to focal symptomatology in these two cases of third ventricle tumour, and will now mention only certain of the more important features.

In the first place, few hypothalamic symptoms were present, which is not surprising when it is remembered that the floor of the third ventricle was not directly involved by the growth in either case. Polyuria and polydipsia, adiposity or loss of weight were absent. Unfortunately, inquiry into sexual potency was omitted. In Case 1, however, transient glycosurea occurred with the first attack of unconsciousness twelve years ago, and in Case 2, excessive sweating and mild fever were present throughout the observed part of the illness. Hyperidrosis has already been described by Penfield [3] in a case of cholesteatoma of the third ventricle, as a manifestation of parasympathetic stimulation.

Abnormal drowsiness, so common with lesions in this region, was a prominent feature in both cases, Fulton and Bailey [2] believe that the neural mechanisms concerned with sleep regulation lie in the posterior part of the hypothalamus close to the midbrain. That the anterior part of the midbrain was affected in each case is shown by

the pupillary abnormalities, including reflex iridoplegia. But since the lethargy appears from the first to have been associated with mental dullness it is safer to assume that it was a general rather than a focal symptom.

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THE AFFERENT PATH OF THE PUPILLARY LIGHT REFLEX IN THE MONKEY.[1]

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MOST explanations of the Argyll Robertson or fixed pupil have accounted for the loss of the pupillary reactivity to light in this syndrome on the basis of a central interruption of the afferent path of the light reflex somewhere along its course to the oculomotor nucleus. A knowledge of the central course of the pathway of the light reflex has, therefore, assumed major importance for an understanding of the Argyll Robertson disorder, and in most of the publications on this subject either clinical or experimental evidence bearing on the localization of this pathway has been presented.

Extensive as this literature has been, it was not until two years ago that satisfactory evidence became available concerning that part of the arc situated within the interior of the brain. The use of a refined technique employing the Horsley-Clarke stereotaxic instrument has made it possible to fill this gap in our knowledge (Ranson and Magoun [1]), and to show that in the cat the path of the pupillary light reflex runs central from the optic tract and the superior quadrigeminal brachium to the pretectal region, or the transition area between the thalamus and superior colliculus. From the pretectal region the pathway swings ventrally around the rostral central grey matter of the aqueduct to the oculomotor nucleus, undergoing a partial decussation in the posterior commissure and another ventral to the central grey matter. It may be emphasized that the pathway was found to include the pretectal region of the brain and not the superior colliculus.

The essential rôle of the pretectal region in the innervation of the pupillary sphincter was next demonstrated in an investigation of the effect upon the light reflex of small, accurately placed lesions made with the aid of the Horsley-Clarke instrument (Magoun and Ranson [2]). In this study it was found that if the pretectal region was almost completely destroyed on both sides of the brain and the rostro-medial portion of the superior colliculus was damaged, the pupillary light reflex

[1] Aided by a grant from the Rockefeller Foundation.

was completely and permanently abolished. Destruction of the superior colliculus alone was found to leave the light reflex intact (Magoun [3]).

The afferent path of the pupillary light reflex has thus been well established in the cat, and it seemed desirable to see if the results of these investigations could be confirmed in the primate brain. The afferent path of the light reflex, as revealed by electrical stimulation, has, therefore, been investigated in the rhesus monkey, *Macaca mulatta*, and the results obtained, which are essentially identical with those determined for the cat, form the subject of this report.

Method.

Stimulation of the brain-stem was performed in a series of eight monkeys, under light nembutal anæsthesia (10-16 mg. per kilo body weight), additional ether being employed during the operative preparation. One animal was a normal monkey. Each of the others had been the object of an earlier investigation of the effect of lesions in the hypothalamus or thalamus, but the presence of a pupillary reaction to light was verified in each animal before experiment, and subsequent microscopical examination of the brain demonstrated that the lesion spared the parts of the optic system concerned in every case.

In the experiments on the light reflex pathway, the skin over the top of the head was incised, 1 to 2 square inches of the vertex of the calvarium was removed, the dura was cut away, and threads were passed through the dura at the margins of the superior sagittal sinus to permit its retraction for midline stimulation. The Horsley-Clarke instrument was adjusted to the animal's head and stimulation of the brain-stem was performed. The present report is concerned exclusively with the pupillo-constrictor responses obtained, and all of these will be described with reference to the left side of the brain.

The use of the Horsley-Clarke stereotaxic instrument has been described in detail by one of us (Ranson, [4]), and the procedures involved in the present series of experiments have been almost identical with those employed in the investigation of the light reflex pathway in the cat (Ranson and Magoun, [1]). For this reason only a brief and general description of the methods need be given here. Use of the Horsley-Clarke instrument makes possible the stimulation by faradization of points within the interior of the brain, at millimetre intervals along electrode punctures, which can be subsequently identified in microscopical sections of the explored area as fine lines of hæmorrhage. By measuring from the base of these punctures, after calculating for

shrinkage, the location of each point of stimulation may be determined, and a correlation made with the response obtained.

In these experiments attention was concentrated upon that portion of the brain-stem made up of the caudal portion of the thalamus, the pretectal region and the superior colliculus. The results from each of the eight animals were first worked up individually and then were plotted on a standard series of seven levels through the area explored, four of which are illustrated in fig. 1.

In stimulation of the lateral portion of the brain-stem, where the optic tract lies in relation to the geniculate bodies and pulvinar, the two electrodes, represented by the exposed tips of otherwise insulated lengths of fine nichrome wire, were separated from one another by a distance of 1 mm. along the axis of the "needle" which consisted of the two wires cemented together. The current was supplied by a single dry cell attached to a Harvard inductorium, the secondary coil of which was set sometimes at 9 cm. and sometimes at 8 cm. from the primary.

In stimulation of the more medial portion of the brain-stem (i.e. the caudal part of the thalamus, the pretectal region, and the superior colliculus from the brachium of the superior colliculus to the midline) the two electrodes were separated from one another by a distance of only 0·2 mm., and the secondary coil of the inductorium was never advanced to less than 9 cm. from the primary, and was often set at 10 cm. Such a weak faradic current, combined with the narrow separation between the electrodes, permitted the stimulation of a strictly localized area surrounding the tips of the electrodes.

Results.

The pupillo-constrictor responses obtained during this series of experiments are seen (fig. 1) to fall into an orderly arrangement with reference to structures within the brain-stem, so that at least from the brachium of the superior colliculus centralward, a connected chain of responses permits the delimitation of the course taken by the light reflex fibres in their central passage to the oculomotor nuclei. It is fortunate that an abundant amount of evidence has been obtained regarding the disputed portion of the light reflex pathway from the brachium of the superior colliculus to the oculomotor nuclei, for less success has met our attempt to trace the non-controverted part of this pathway from the optic tract to the brachium of the superior colliculus.

Optic tract.—Difficulty was encountered in activating the light reflex pathway lateral to the brachium of the superior colliculus in the

monkey, and even with wide-tipped electrodes and a rather strong faradic current only a small number of responses were obtained. These consisted of a slight bilateral constriction, equal in the ipsi- and contralateral pupils.

The localization of these few responses suggests that the light reflex fibres pass medially from the optic tract in the region of the lateral geniculate body (fig. 1, A) into the band of fibres which runs caudally along the lateral aspect of the medial geniculate body (fig. 1, B), and which then turns dorsally to enter the brachium of the superior colliculus (fig. 1, C and D). This small amount of evidence can do no more than suggest such a course, which, it may be noted, is comparable to that established for the cat (Ranson and Magoun [1]). No evidence has been obtained in favour of the possibility that light reflex fibres are contained in the broad tract of optic fibres which passes lateral and dorsal to the lateral geniculate body, where it joins with a prominent bundle of fibres which may represent cortical connections, from which also, no pupillo-constrictor responses have been obtained in these experiments (fig. 1, A and B).

We are unable to explain the difficulty encountered in activating the light reflex fibres in this lateral portion of the brain-stem in the monkey. It may be that the fibres take a scattered course, but even where they appear to become condensed into fairly compact strands, as on the lateral aspect of the medial geniculate body (fig. 1, B) or in the brachium of the superior colliculus (fig. 1, C and D), only slight constrictor responses have been obtained by stimulation. Stimulation of the retinal fibres in the compact optic tract peripheral to the lateral geniculate body, which was not attempted in these experiments, might be expected to yield a maximal pupillo-constriction.

Brachium of the superior colliculus.—A sufficient number of pupillo-constrictor reactions have been obtained from stimulation of the brachium of the superior colliculus in these experiments to indicate that in the monkey the afferent fibres of the pupillary light reflex reach the more medial portion of the brain-stem by way of this structure (confirming Karplus and Kreidl [5]), just as they have been demonstrated to do in the cat (Karplus and Kreidl [5]; Ranson and Magoun [1]). In the monkey, stimulation of the brachium of the superior colliculus has produced a slight bilateral constriction, equal in the ipsi- and contralateral pupils. It is perfectly clear from these experiments, however, that after reaching the brachium of the superior colliculus, the afferent light reflex fibres *do not* turn medially and caudally to enter the substance of the superior colliculus itself.

Superior colliculus.—Repeated stimulation of the superior colliculus (fig. 1, C and D) in each of the experimental animals has always failed to yield pupillo-constrictor reactions. With the more localized stimulation employed in these experiments it has been found that the pupillo-dilator responses encountered by other workers (Ferrier [6]; Karplus and Kreidl [5]) from electrical stimulation of the superior colliculus in the

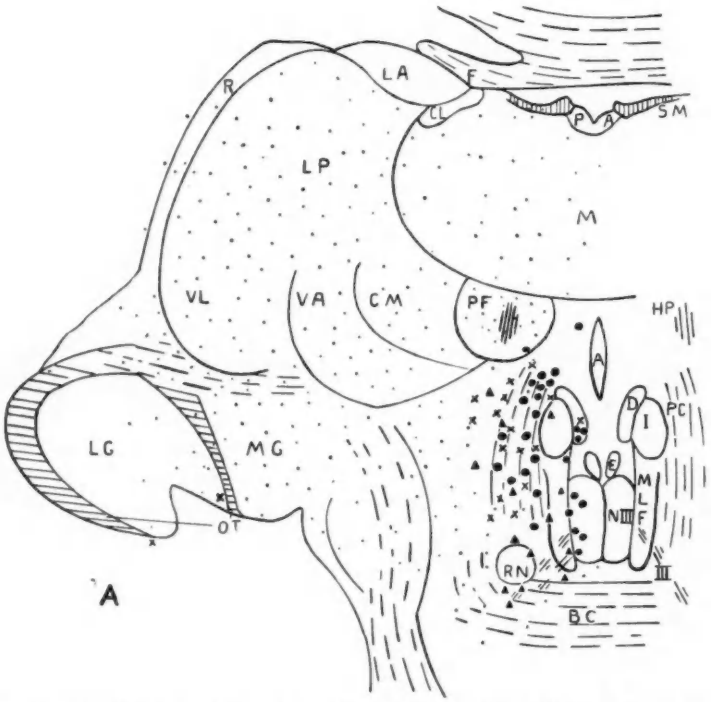
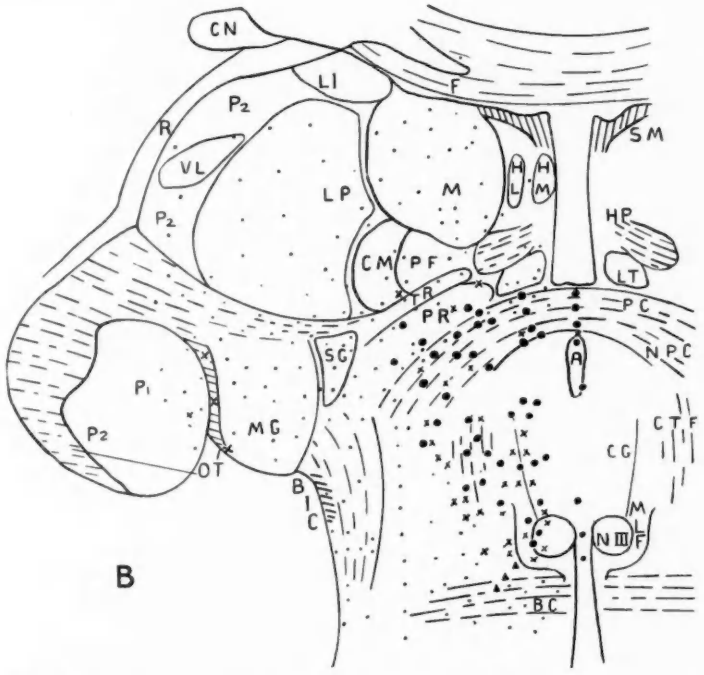


FIG. 1.—The situations of the points, stimulation of which yielded pupillo-constrictor responses in these experiments, are shown here in four levels through the left side of the brain-stem of the monkey. These levels are in the vertical plane of the Horsley-Clarke instrument, and extend serially and caudally from the caudal portion of the thalamus (A), through the pretectal region (B), to the superior colliculus (C and D). The positions in these levels of the points whose stimulation produced marked bilateral constriction are indicated by solid circles (●); the locations of points yielding slight bilateral constrictor responses

monkey, are not observed until the electrodes have been lowered through the tectum into the underlying tegmentum of the midbrain or central grey matter surrounding the aqueduct. The absence of pupillo-constrictor reactions on stimulation of the superior colliculus in the monkey is in complete agreement with the results obtained from the cat

(Ranson and Magoun [1]), and it may now be shown that in the monkey, just as in the cat, the light reflex fibres after reaching the brachium of the superior colliculus turn rostrally and medially to enter the pretectal region.

Pretectal region.—The pretectal region (figs. 1, B and 2) forms a zone of transition between the thalamus and superior colliculus, and a detailed



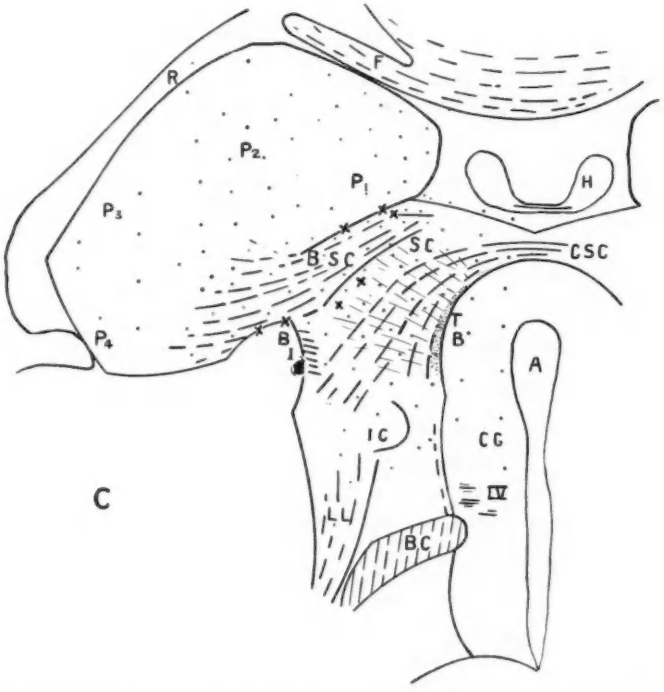
are indicated by crosses (×); and the positions of points yielding ipsilateral constrictor responses are shown by solid triangles (▲). Dots indicate the points, stimulation of which did not produce a pupillo-constrictor reaction. Level A illustrates data from 8 animals, B from 7 animals, C from 3 animals and D from 4 animals. Abbreviations for Fig. 1 are as follows:—
A, aqueduct; BC, brachium conjunctivum; BIC, brachium inferior colliculus; BSC, brachium superior colliculus: CN, caudate nucleus; CG, central grey matter; CL, nucleus centralis lateralis; CM, centre médian; CSC, commissure of superior colliculus; CTF,

anatomical description of its configuration in the monkey is presented elsewhere (Atlas [7]).

In marked contrast to the inexcitability of the superior colliculus (fig. 1, C and D), stimulation of the pretectal region in the monkey (fig. 1, B), as in the cat (Ranson aud Magoun [1]), has yielded a large

number of pupillo-constrictor responses. These reactions in the monkey have consisted of a slight to marked bilateral constriction, equal in the ipsi- and contralateral pupils. They represent a rostro-medial continuation of the constrictor responses elicited from the brachium of the superior colliculus (fig. 1, c).

In the cat it has been shown that typical constrictor reactions are

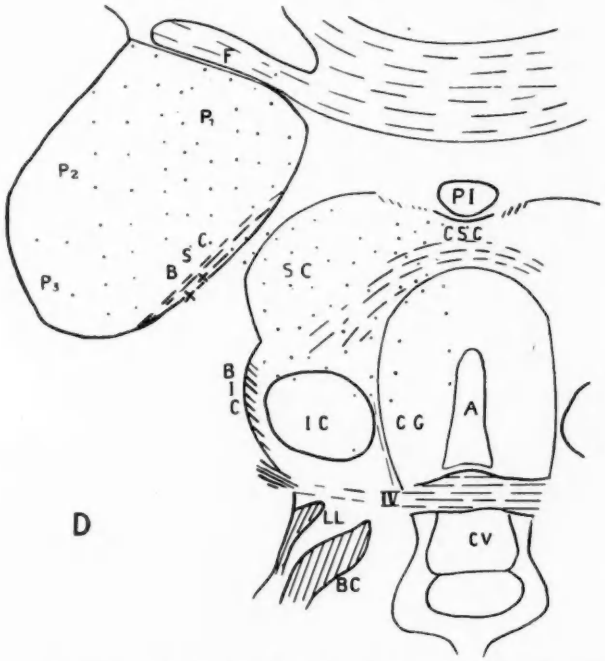


central tegmental fasciculus; CV, cerebellar vermis; D, nucleus of Darkschewitsch; E, nucleus of Edinger-Westphal; F, fornix; H, habenula; HL, lateral habenular nucleus; HM, medial habenular nucleus; HP, habenulo-peduncular tract; I, interstitial nucleus; IC, inferior colliculus; LA, nucleus lateralis pars anterior; LI, nucleus lateralis pars intermedia; LP, nucleus lateralis pars posterior; LG, lateral geniculate body; LL, lateral lemniscus; LT, nucleus lentiformis thalami; MG, medial geniculate body; M, medial

still obtainable from the pretectal region after the complete degeneration of all primary optic fibres (Hare, Magoun, and Ranson [8], and they are produced, therefore, by the activation of secondary neurones in the light reflex arc. Stated in other words, the pretectal region has been demonstrated in the cat to represent the central end-station of the

primary optic fibres concerned in the pupillary light reflex. We see no reason to doubt the probability that this holds true also in the monkey.

The excitable area in the monkey (fig. 1, B and A) includes, in addition to the pretectal region, the posterior commissure and a broad arch around the central grey matter. This may be taken as evidence that of the fibres arising from the secondary optic neurones in the pre-



dorsal nucleus; MLF, medial longitudinal fasciculus; NPC, nucleus posterior commissure; N III, oculomotor nucleus; OT, optic tract; PA, nucleus paraventricularis; PC, posterior commissure; PF, nucleus parafascicularis; PI, pineal body; PR, pretectal nucleus; P 1, 2, 3, 4, pulvinar parts 1, 2, 3, 4; R, nucleus reticularis; RN, red nucleus; SC, superior colliculus; SG, nucleus suprageniculatus; SM, stria medullaris; TB, tectobulbar fibres; TR, nucleus of the optic tract; VA, nucleus ventralis pars arcuata; VL, nucleus ventralis lateralis; III, oculomotor nerve; IV, trochlear nerve.

tectal region, some cross to the opposite side of the brain in the posterior commissure, and others descend directly around the central grey matter to the oculomotor nuclei.

Posterior commissure. — Electrical stimulation of the posterior commissure in the monkey (fig. 2) has yielded a large number of

slight to marked constrictor responses, equal in the two pupils. These reactions have been obtained from fibres of the posterior commissure both in the midline (fig. 1, B), and on one side of the brain (fig. 1, B

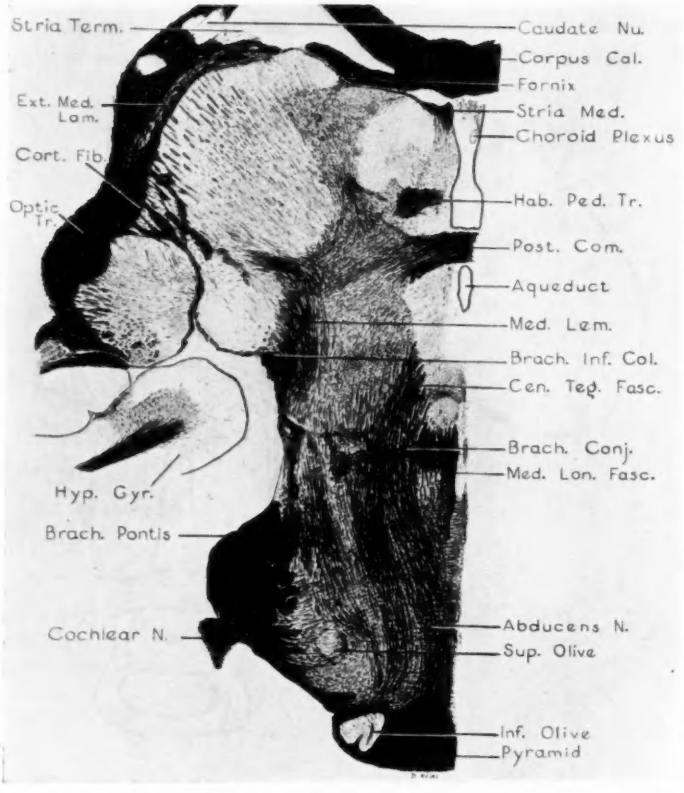


FIG. 2.—Drawing of a section stained by Weil's method through the pretectal region of the brain-stem of the monkey at the level shown in Fig. 1, B. This section is in the vertical plane of the Horsley-Clarke co-ordinates which is transverse through the thalamus, but becomes oblique through the midbrain because of the angle given to this portion of the brain stem by the cephalic flexure. The figure serves both to orient the reader and to illustrate the relationships of the myelinated fibres of the pretectal region. × 4·5. Abbreviations for Fig. 2 are as follows:—

Abducens N., abducens nerve; Brach. Inf. Col., brachium of the inferior colliculus; Brach. Conj., brachium conjunctivum; Brach. Pontis, brachium pontis; Caudate Nu., caudate nucleus; Cen. Teg. Fasc., central tegmental fasciculus; Cochlear N., cochlear nerve; Cort. Fib., cortical fibres; Corpus Cal., corpus callosum; Ext. Med. Lam., external medullary lamina; Hab. Ped. Tr., habenulo-peduncular tract; Hyp. Gyr., hippocampal gyrus; Inf. Olive, inferior olive; Med. Lem., medial lemniscus; Med. Lon. Fasc., medial longitudinal fasciculus; Optic Tr., optic tract; Post. Com., posterior commissure; Stria Med., stria medullaris; Stria Term., stria terminalis; Sup. Olive, superior olive.

and A). They unquestionably demonstrate a crossing of fibres concerned with the pupillary light reflex in the posterior commissure of the monkey, as has been previously demonstrated in the cat (Ranson and Magoun [1]).

There is no way of determining from these stimulation experiments the quantitative amount of this central decussation in the light reflex path. In the cat it has been shown to be only partial, for destruction of the posterior commissure in the midline has led to a reduction, but not to a complete abolition of the light reflex (Spiegel [9]; Magoun, Ranson and Mayer [10]; Scala and Spiegel [11]). This observation should not be applied directly to the monkey, however, for Karplus and Kreidl [5] have established the fact that the crossing of the light reflex path in the optic chiasma differs quantitatively in the cat and monkey, and the same may hold true for central decussations in this pathway.

Fibres descending around the rostral end of the central grey matter of the aqueduct.—From the pretectal region and posterior commissure of the monkey a large number of pupillo-constrictor responses may be traced ventrally around the rostral pole of the central grey matter of the aqueduct to the oculomotor nuclei (fig. 1, A and B). Some of the reactive points are situated among the descending fibres of the posterior commissure proper (fig. 1, A), while others are located in the lateral portion of the central grey matter and the adjacent fibre systems of the rostral part of the mesencephalic tegmentum (fig. 1, B.) All of the reactive points are situated rostral to the tecto-bulbar fibres (fig. 1, C). In our opinion these responses represent the activation of fibres from the pretectal region of the opposite side of the brain which have crossed in the posterior commissure, and of fibres from the pretectal region of the same side, both of which groups of fibres descend through this area to the oculomotor nuclei. Stimulation of these fibres yields a bilateral response, consisting of a slight to marked constriction, which in the larger number of cases is equal in the ipsi- and contralateral pupils, but which in some instances is greater in the pupil of the same side. In a few responses the pupil of the same side only contracted, the contralateral pupil either remaining inactive or dilating.

Ventral decussation.—The fact that bilateral pupillo-constrictor responses are obtained from stimulation of points on the lateral aspect of the central grey matter between the posterior commissure and the oculomotor nuclei (fig. 1, A and B), shows that in the monkey, as in the cat (Ranson and Magoun [1], further crossing in the light reflex

pathway takes place ventral to the cerebral aqueduct. This ventral decussation could occur in the afferent path just before its synapse with the oculomotor neurones. It could also consist of inter-oculomotor connections, or of crossed efferent oculomotor fibres; or, of course, of combinations of these possibilities. The crossing would appear to be in the immediate vicinity of the oculomotor nucleus, for stimulation of points through the dorsal tegmentum up to the periphery of the oculomotor nucleus has yielded bilateral pupillo-constriction, while excitation of the root fibres of the oculomotor nerve just below the nucleus, has yielded a constriction of the ipsilateral pupil alone (fig. 1, A and B).

The impression might be gained from our first diagrammatic reconstruction of the light reflex pathway in the cat ([1], fig. 3, p. 1201), that this ventral decussation was an internuclear one, although it was stated in the accompanying text that we were unable to say whether it represented a crossing of secondary optic or of oculomotor fibres. In two subsequent publications [2, 10], this diagram has been altered to represent the ventral decussation as being made up of afferent light reflex fibres.

A comparison of the data from the monkey with those from the cat, indicates that this suggested ventral decussation is greater in the monkey than in the cat. Stimulation in the cat [12] of 141 points on one side of the central grey matter, between the posterior commissure and the oculomotor nuclei, caused constriction which was greater in the ipsilateral pupil in 88, or in about two-thirds of the responses, and in 45 of these the contralateral pupil showed no constriction whatever. In the cat constriction was equal in the two pupils in 53, or in about one-third of the reactions.

In the responses from stimulation of 118 similarly situated points in the monkey (present investigation), constriction was greater in the ipsilateral pupil in only 22, or about one-fifth of the responses, and in only 9 of these did the contralateral pupil fail to contract at all. In the reactions from the monkey, constriction was equal in the two pupils in 96, or in about four-fifths of the responses.

The results of the present investigation in the monkey do not permit the more specific localization of this presumed ventrally situated crossing of the light reflex pathway, nor do they enable us to add any information to the problem of the localization of the pupillo-constrictor component within the oculomotor nucleus itself.

Discussion.

The present investigation of the path of the pupillary light reflex within the brain-stem of the monkey indicates that the light reflex fibres traverse the portion of the optic tract which runs medial to the lateral geniculate body, and passes caudally along the lateral and dorsal aspects of the medial geniculate body to reach the brachium of the superior colliculus. It is clear that after reaching the brachium of the superior colliculus the light reflex pathway does not enter the superior colliculus itself, but turns rostrally and medially into the pretectal region. From the pretectal region the pathway descends around the rostral end of the central grey matter of the aqueduct to the oculomotor nuclei. Central crossings in the path were found to occur both in the posterior commissure and ventral to the cerebral aqueduct in the immediate vicinity of the oculomotor nuclei. The course of the light reflex pathway within the brain-stem of the monkey has thus been found to be essentially identical with that previously demonstrated for the cat (Ranson and Magoun [1].

The results of our investigation of the light reflex pathway [1] have recently been vigorously criticized by Harris [13] who has suggested, on very insufficient evidence, an alternative pathway for the pupillary light reflex. In Harris' opinion the superior colliculus and tecto-bulbar fibres form the central link in the light reflex path, and this is a view which has been widely held in spite of the large amount of opposing evidence.

If the superior colliculus forms an essential part of the light reflex pathway it might be expected, first, that stimulation of the superior colliculus should cause a pupillo-constrictor response ; and secondly, that destruction of the superior colliculus should abolish the light reflex. The reverse of these two considerations has been repeatedly demonstrated.

Stimulation of the dorsal surface of the superior colliculus has been found to produce a dilatation of the pupil by Knoll [14] in the rabbit ; by Ferrier [6] in the rabbit, cat, jackal, dog and monkey, and by Karplus and Kreidl [5] in the cat and monkey. Our results in the cat [1] and in the monkey (see above) show that with a more localized stimulus no response from the pupil is obtained until the electrodes pass through the superior colliculus and reach the underlying central grey matter of the aqueduct or the tegmentum of the midbrain, where stimulation causes pupillo-dilatation. It is clear that stimulation of the superior colliculus does not produce a constriction of the pupil.

Furthermore, the light reflex has been found to remain intact after destruction of the superior colliculus by Knoll [14] in the rabbit, Bechterew [15] in the dog and pigeon (corpora bigemina), Gudden [16] in the rabbit, Ferrier and Turner [17] in the monkey, Levinsohn [18] in the rabbit and monkey, Bernheimer [19] in the monkey, Keller and Stewart [20] and one of us (Magoun [3]) in the cat. It is, therefore, clear that destruction of the superior colliculus leaves the light reflex intact.

Stimuli have been applied many times to the tecto-bulbar fibres in their passage through the mesencephalic tegmentum in both the cat and monkey in investigations in this laboratory, and such stimuli have never produced pupillo-constrictor responses. In this connection attention should be called to the important observation of Rasmussen [21] that in the cat efferent fibres from the superior colliculus do not terminate in the oculomotor nuclei, the tecto-fugal system being distributed chiefly to the substantia nigra, pons, the reticular formation, and to the motor nuclei of the lower portion of the medulla oblongata and the cervical segments of the spinal cord.

It is difficult to understand how, in the face of this overwhelming amount of opposing evidence, the superior colliculus and its efferent connections can still be considered the central link in the pathway for the pupillary reaction to light.

Further objection to the results of our investigation of the light reflex pathway in the cat [1] has been made by Harris [13] from the point of view of the central crossing of this path in the posterior commissure. In two experiments reported in 1904, Harris [22] observed the effect of section of the posterior commissure on the light reflex of the cat. Following operation there was a complete loss of the light reaction in his animals lasting in one case for three days. In both cases, however, the pupillary reactions returned and became, according to Harris, " quite normal."

Several factors make it difficult to estimate the value of these observations. No mention is made of the histological verification of the extent of the lesion, and without this we cannot be sure that the posterior commissure was completely divided. We have no way of knowing what a " normal " light reaction meant to Harris at the time his experiments were performed, for no measurements of the pupillary changes are given. Finally, it is impossible to learn when this return to " normal " took place, for no day-by-day records or survival times of the animals are included.

The more rigid standards employed in three recent studies of this problem have shown that while the pupillary light reflex is not abolished after complete and histologically verified destruction of the posterior commissure in the cat (Spiegel [9]; Magoun, Ranson, and Mayer [10]; Scala and Spiegel [11]), it suffers an impairment, apparent as an easily measured reduction in the amount of pupillo-constriction to a standard source of light; the five animals employed in our investigation were studied over periods of from one to four weeks after operation.

The data from stimulation experiments concur with the results obtained from these recent studies of the effects of experimental lesions in indicating that some crossing of the light reflex path takes place in the posterior commissure, for bilateral pupillo-constriction has been obtained from localized stimulation of the posterior commissure both in the cat [1, 8] and in the monkey (see above). There is no foundation for the unusual suggestion [13] that these effects are produced by a spread of current to the oculomotor nuclei over the habenulo-peduncular tract; if for no other reason, because stimulation of the habenulo-peduncular tract itself yields no response either in the cat or monkey, and effects cannot be explained as due to a spread of current to a structure which gives no response when activated directly.

It is clear, both from stimulation experiments and from critical studies of the effect of lesions in experimental animals, that some crossing of the pupillary light reflex pathway takes place in the posterior commissure. Two recent clinical studies have gone even further in ascribing a complete loss of the pupillary light reflex to destruction of the posterior commissure in man (Collier, [23]; Herrmann, [24]).

The final objection made by Harris [13] to our work [1] is related to the occurrence of a synapse in the afferent path of the light reflex. It is true that synapses were omitted in our diagrammatic reconstruction of the light reflex path (Ranson and Magoun [1], fig. 3, p. 1201), although in the description of this figure attention was called to this omission. Synapses were not shown in this diagram because at that time their location was unknown. This location has since been determined, for the first time, by stimulation of the brain-stem of the cat after the complete degeneration of all primary optic fibres, as a result of bilateral intracranial section of the optic nerves (Hare, Magoun and Ranson, [8]). The results of this investigation demonstrate that a synapse in the afferent path of the light reflex is situated in the pretectal region.

Other information pointing to the existence of this synapse, together

with the importance of the central decussation in the light reflex path for an understanding of the consensual pupillary reaction, have already been considered in our review on the light reflex pathway [25]. We can find no justification for the imputation [13] that the results of our work do not account for the presence of a consensual pupillary reaction, either under normal conditions or after bitemporal hemianopsia (mid-sagittal section of the optic chiasma).

SUMMARY.

An investigation of the afferent path of the pupillary light reflex in the monkey by electrical stimulation of the interior of the brain-stem with the aid of the Horsley-Clarke instrument, indicates that the light reflex fibres traverse the portion of the optic tract which runs medial to the lateral geniculate body, and passes caudally along the lateral and dorsal aspects of the medial geniculate body, to reach the brachium of the superior colliculus. It is clear that after reaching the brachium of the superior colliculus the light reflex pathway does not enter the superior colliculus itself, but turns rostrally and medially into the pretectal region, or the transition area between thalamus and midbrain. From the pretectal region the pathway descends around the rostral end of the central grey matter of the aqueduct to the oculomotor nuclei. Central crossings in the path were found to occur both in the posterior commissure and ventral to the cerebral aqueduct in the immediate vicinity of the oculomotor nuclei.

The afferent path of the light reflex in the monkey has thus been found to be essentially identical with that recently established for the cat.

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CLINICAL APPLICATION OF THE CRANIO-VERTEBRAL DYNAMICS TO ENCEPHALOGRAPHY.[1]

BY THEODORE J. C. VON STORCH.

"Wherefore, in order to form a just notion of the body of man, it ought to be considered as a hydraulic machine contrived with the most exquisite art, in which there are numberless tubes properly adjusted and disposed for the conveyance of fluids of different kinds."—RICHARD MEAD, "Medical Precepts and Cautions," London, 1751.

PART I.—THEORY.

I. INTRODUCTION.—The replacement of cerebrospinal fluid by gas for diagnostic or therapeutic purposes has become an increasingly valuable neurological procedure since its introduction by Dandy [1, 2] in 1918. There are three types of subarachnoid pneumography. Myelography is the roentgenographic demonstration of the vertebral subarachnoid spaces subsequent to the replacement of cerebrospinal fluid by gas [3]. Ventriculography consists of roentgenographic demonstration of the cerebral ventricles following the direct intraventricular introduction of gas. Encephalography is the roentgenographic demonstration of the cerebral ventriculo-subarachnoid spaces following replacement of the cerebrospinal fluid by gas through cisternal or lumbar punctures.

Following the work of Bingel [4 to 9] and Wideroe [10] in 1921, lumbar encephalography has become increasingly valuable as a diagnostic and therapeutic procedure. This study is concerned solely with lumbar encephalography.

Lumbar encephalography is chiefly a diagnostic procedure, but it is occasionally used for therapeutic purposes. Its primary purpose is to make visible the intracranial ventriculo-subarachnoid spaces. Replacement of fluid by a gas is conducted through one or two needles inserted into the subarachnoid space at about the level of the fourth lumbar vertebra. Inferences may be drawn from the roentgenograms as to the condition of the brain, nerves, blood-vessels and other structures outlined by the gas. It is, therefore, of prime importance that the

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ventriculo-subarachnoid spaces be filled with gas in such fashion as to produce the minimum distortion of cerebral structures and at the same time a minimum reaction on the part of the patient.

II. LITERATURE.—Any consideration of encephalography depends upon an understanding of the fundamental dynamic changes occurring in the subarachnoid space during encephalography. A review of the literature reveals only two studies on the subarachnoid dynamics of encephalography. Neither of these was based upon direct observations in man, but they were considerations of the probable sequence of events.

Castex and Ontaneda in 1932 [11 to 13] published several articles on the lumbar, cisternal and ventricular spinal fluid pressures following the replacement of cerebrospinal fluid by air. They demonstrated these changes in a glass and rubber model of the cranio-vertebral system. The apparatus used consisted of an inverted glass flask representing the cranium. A thin rubber sac representing the vertebral subarachnoid space was hung from the neck of the flask and the model was filled with water in such fashion that when erect the lumbar, cisternal and ventricular pressures accepted for man were reproduced at the proper levels. It was then demonstrated that a progressive replacement of water by equal volumes of air resulted in a decrease of the "lumbar pressure" and an increase of "cisternal and ventricular pressures" until, when the water was entirely replaced, all three pressures became the same. It was then demonstrated that if the "lumbar pressure" was maintained at its original level of 400 mm. of water[1] by replacement of the water with a larger volume of air, the other pressures gradually increased until again all three were equal, but this time at a pressure of 400 mm.

The model used was essentially that described by Propping in 1908 [14] and is too simplified to be strictly applicable to the cranio-vertebral system of man. The authors have, however, demonstrated two phenomena of cardinal importance to the practice of encephalography. The first is that during the replacement of spinal fluid by equal volumes of gas the lumbar pressure decreases, while the cisternal and ventricular pressures increase until, when completed, all become equal

[1] Hereafter, unless otherwise indicated, pressure or force per unit area will be expressed in the normal clinical units, millimetres of water, that is that pressure which will support a column of so many millimetres of water against atmospheric pressure or 760 mm. Hg (equal to 14·6 lb per sq. in.). For example 136 mm. of water = 10 mm. of mercury = 0·19 lb. per sq. in. pressure.

at a level approximating zero (atmospheric) pressure. The second is that maintenance of the original lumbar pressure during encephalography would produce a dangerously increased intracranial pressure of about 400 mm. of water in the ventricles.

Schube, in 1934 [15], published the other study dealing with the physical dynamics of encephalography. It is essentially a detailed derivation of the physical formulæ upon which is based the practice of replacing any given volume of cerebrospinal fluid by a smaller volume of gas. The formulæ are accurate, and Charles's law cannot be denied (i.e. that the pressure exerted by a given volume of gas varies directly with the temperature of the gas if the volume be kept constant). This law, however, applies to rigid containers and does not apply to pressure if the gas be allowed to change its volume. The accurate conception of the cerebrospinal fluid system is that it is not enclosed in a rigid container, but is subject to semi-rigid boundaries [16 to 26]. The author postulates that "the encephalographic procedure, therefore, consists of removing a definite volume of fluid under a relatively constant temperature and pressure *from an area with rigid boundaries* (the skull and spinal canal) and replacing it simultaneously with an equal volume of air under the same or different pressure and markedly different temperature. Hence, starting with this erroneous postulate, the author demonstrates that if room-air were made to replace an equal volume of cerebrospinal fluid, the increase in temperature would increase the pressure by 0·8 lb. per sq. in. The use of purely physical terminology has been unfortunate, as it unwittingly conceals an otherwise obvious error. This increase of 0·8 lb. per sq. in. is equal to 41·5 mm. of mercury or 564 mm. of water as expressed in clinical units. Thus an initial spinal-fluid pressure of 450 mm. of water, as measured in the sitting position at the lumbar sac, would be increased to 1,014·4 mm. of water by replacement of *any* volume of cerebrospinal fluid by an equal volume of air at room temperature. This, of course, does not occur. No such observations were made by the author during encephalography, nor have any such observations been reported in the literature. It is unfortunate that the physical arguments presented might appear to justify the old axiomatic method of replacing cerebrospinal fluid by smaller volumes of gas. One of the primary concerns of this paper is an attempt to demonstrate conclusively the error of such a procedure.

III. Observations.—In order to gather further evidence concerning the physiology of encephalography, experiments were conducted in connection with the encephalograms obtained in this clinic from 1932

to 1935. During this period 119 patients were investigated by the author or under his direct supervision. Each encephalogram was undertaken as a diagnostic measure, but the technique varied according to the circumstances. A preliminary report has been published [31].

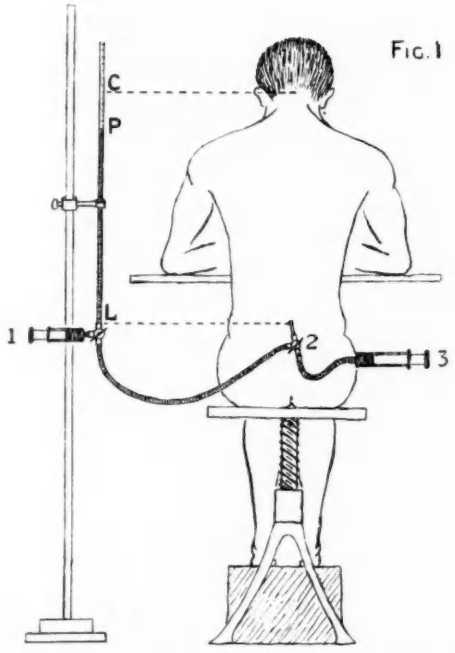

FIG. 1.—*A Method for Alternate Replacement Encephalography.* The patient sits or is supported with back anteroflexed. An Ayer-Fremont-Smith needle with attached three-way stopcock (2) is used for the lumbar puncture. Previous to measurement of pressures the 2 mm. bore open-end water manometer (C–P–L) and attached rubber tubing (L–2) is filled with sterile saline solution by the syringe (1) via the three-way stopcock at (L). With the zero level of the manometer (L) on a level with the lumbar puncture needle the meniscus in the manometer is adjusted to about level (C), and the stopcock opened to (2). Stopcock (2) is then opened to the subarachnoid space and the lumbar cerebrospinal fluid pressure is recorded. Stopcock (2) is now opened to the syringe (3) and 5 to 20 c.c. of cerebrospinal fluid allowed to flow into the syringe. Stopcock (2) is again connected with (1) and the "removal pressure" measured while the syringe (3) is disconnected and emptied into a graduate. With the syringe (3) 5 to 20 c.c. of room air is then introduced into the subarachnoid space via (2), the "replacement pressure" recorded, and the process continued until air returns into the syringe. (L) designates the lumbar, and (C) the cisternal level, while (L–P) indicates the lumbar cerebrospinal fluid pressure.

(1) *Relationship of the recumbent to the sitting cerebrospinal fluid pressure.*—In 105 cases the cerebrospinal fluid pressure was measured several days before encephalography. Lumbar puncture was performed

with the usual aseptic technique using a Fremont-Smith needle with attached three-way stopcock. Pressures were measured in millimetres of water with the Ayer-Fremont-Smith spinal fluid manometer attached directly to the needle. Throughout all the investigations the greatest care was taken to measure the pressures accurately, without loss of spinal fluid, and under standard conditions. These pressures, measured with the patient recumbent, ranged from 50 to 250 and averaged 131 mm. of water. In another series of sixty cases the lumbar cerebrospinal fluid pressure was measured immediately prior to encephalography while the patient was recumbent. The previous technique was employed except that in some cases the manometer was filled with sterile saline solution and attached to the needle by rubber tubing which was also filled with saline solution (fig. 1). These pressures ranged from 70 to 250 averaging 134 mm. of water. One hundred and six patients were then placed in the sitting position and the pressures again recorded. These latter pressures ranged from 145 to 550 averaging 371 mm. of water.

In both series it was noted that the lumbar spinal fluid pressure measured with the patient in the sitting position (371) was always greater than the pressure in the recumbent position (131). In no case was there any other relationship between the two pressures. That is, patients with low pressures when lying down might have either low or high pressures when erect, and patients with high pressures when lying down might also have either low or high pressures when sitting erect. The pressures in the sitting position appeared to bear no direct relationship to age or size of the patient. As a rule the lower pressures were found in the age group below 15, but several in this group exhibited high pressures and several low pressures were exhibited by adults.

(2) *Relation of the cisterno-lumbar distance to the lumbar cerebrospinal fluid pressure with the patient erect.*—In 100 cases the vertical distance between the lumbar puncture needle (at the fourth lumbar space) and the cisterna magna (fig. 1) was measured as well as the lumbar cerebrospinal fluid pressures of the patient when recumbent and sitting. This distance ranged from 200 to 500 and averaged 397 mm. There was a definite relation between the lumbar spinal fluid pressure in the erect position and the cisterno-lumbar distance. The difference between the two averaged only 50 mm., but ranged from minus 150 to plus 250 mm. In fifteen cases the vertical distance was less than the lumbar pressure, the difference ranging from 20 to

150 mm. and averaging 71 mm. In twenty-seven cases the distance and pressure were equal. In the majority, fifty-eight cases, the vertical distance was greater than the lumbar pressure by an average of 68 mm., the range being from 10 to 205 mm.

It should be noted that all the patients were somewhat antero-flexed so that the vertical distance measured was less than the anatomical lumbo-cisternal distance. The patients ranged in age from nine months to 64 years and there was a wide variation in length of back.

(3) *Pressure changes caused by replacement of cerebrospinal fluid by air.*—In twenty cases measurements of the lumbar cerebrospinal fluid pressure were taken after the withdrawal of each sample of cerebrospinal fluid and after the replacement of each volume of air, during the course of the entire encephalogram. The method is outlined in fig. 1. Room air was introduced slowly by syringe after withdrawal of cerebrospinal fluid. Precautions were taken at all times during the experiments to prevent leakage of air on its passage into the subarachnoid space. The apparatus was tested at frequent intervals during each encephalogram to insure that all connections were air tight. The ratio of air introduced to fluid removed (A/F) varied with each step in replacement. The total volumes replaced varied in each case, but all available cerebrospinal fluid was removed before replacement was considered complete.

In thirty-one cases of the entire series (119) an average of 13 c.c. less air was introduced (A/F less than 1); in twenty-four cases equal volumes of air replaced fluid (A/F equal to 1); and in sixty-four cases an average of 18 c.c. more air replaced fluid (A/F greater than 1) (fig. 2). The range when A/F was less than 1 was 45 c.c. to 5 c.c. less air. The range when A/F was greater than 1 was 5 c.c. to 40 c.c. more air.

In all cases the lumbar cerebrospinal fluid pressure decreased when each sample of fluid was removed and increased when air was replaced. The amplitude of these pressure changes was directly proportional to the volume of fluid removed and air replacing it at each step. In all cases, regardless of the value of A/F, the lumbar cerebrospinal fluid pressure gradually approached atmospheric pressure (fig. 2). In order to maintain a positive pressure in the lumbar subarachnoid space at completion of encephalography it was usually necessary to introduce a volume of air greater than the volume of fluid removed (A/F greater than 1). The value of A/F necessary to maintain an adequate subarachnoid pressure was an unpredictable individual variant, although it was greater than unity in the majority of cases. It was noted that

when the initial spinal fluid pressure with the patient erect was low, it was necessary to increase the ratio A/F in order to maintain an adequate final subarachnoid pressure.

(4) *Effect of positional alterations on the gas-filled subarachnoid space.*—This "adequate" final lumbar subarachnoid pressure, after complete replacement of cerebrospinal fluid by air, was considered to be a pressure about equal to the previously measured lumbar cerebrospinal fluid pressure for that particular patient when recumbent. In a series of 101 cases, the lumbar subarachnoid pressures were measured with the patient sitting up after complete replacement of spinal fluid. These

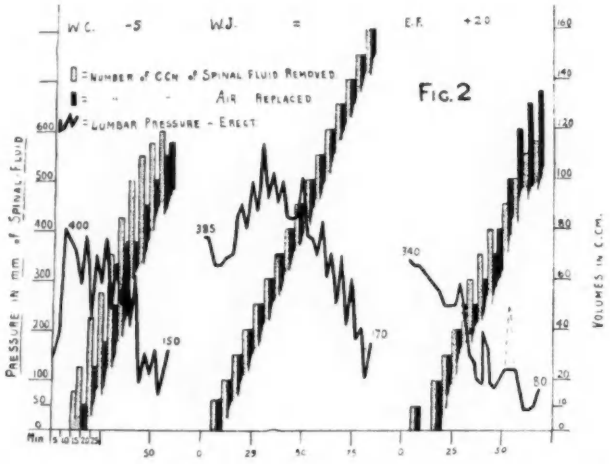

FIG. 2.—*Lumbar Cerebrospinal Fluid Pressures in Man during Lumbar Encephalography in the Sitting Position.* Three typical cases are figured showing the variations in type of curves. It will be noted that in all types the pressure irregularly but progressively decreases regardless of the relative or total volumes of fluid replaced by air.

pressures ranged from 0 to 390, averaging 156 mm. of water. Eighty-one patients were then placed in the lateral recumbent position and the lumbar pressures measured again. The latter pressures ranged from 0 to 350, averaging 140 mm. of water. The difference between the "final sitting" and "final recumbent" pressures was calculated. It averaged 11 mm., ranging from 0 to 100 mm. of water. In no individual case was the pressure of the subject in the recumbent position after encephalography greater than the pressure in the erect position. Thus the patient sitting upright was left with a final subarachnoid pressure at the lumbar sac approximately equal to his initial pressure when lying

down. This latter pressure was, therefore, present when he was returned to bed. As noted, it was necessary in the great majority of cases to introduce more air than the volume of fluid removed in order to maintain such a pressure.

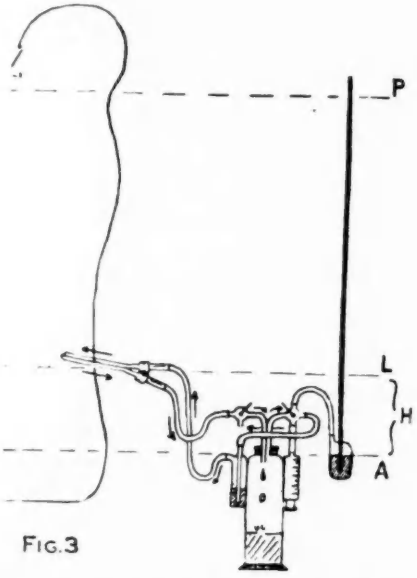

FIG. 3

FIG. 3.—*An Automatic Simultaneous Replacement Apparatus for Encephalography.* Cerebrospinal fluid flows through one bore of a double needle into a closed container, displacing air from it through a sterile water bath, into the lumbar subarachnoid via the second bore of the needle. The pressure in the bottle is continuously measured by an open-end barometric type water manometer (A L P). As the bottle is placed a distance H below the lumbar needle, the pressure in the bottle A P, = L P + H. Therefore, the lumbar subarachnoid pressure L P, = A P − H when the apparatus is in equilibrium (when flow has ceased). While running the pressure A P approximates L P dependent upon the rate of flow. Thus the lumbar spinal fluid pressure may be observed at all times. By use of the syringe the volume and type of gas may be controlled as well as the pressure in the bottle or subarachnoid. After the initial adjustment replacement is automatic and alternations of pressure do not occur.

(5) *Use of an automatic simultaneous replacement method.*—As soon as the principle of replacement by pressure was conceived an apparatus[1] was devised to facilitate the technique. This apparatus is described in detail elsewhere [32]. It consists of a closed container connected with a syringe, manometer and sterile water-bath in such

[1] Manufactured by Macalster Bicknell Company, 171, Washington Street, Cambridge, Mass.

fashion as to control the type, volume, temperature, pressure and rate of flow of the gas as well as the pressure, volume, and rate of flow of the cerebrospinal fluid (fig. 3). The apparatus was operated automatically and successfully in thirty-nine cases. When fluid was replaced in this manner the alternate decrease and increase in subarachnoid pressure was prevented (fig. 4). In the cases in which the apparatus was used

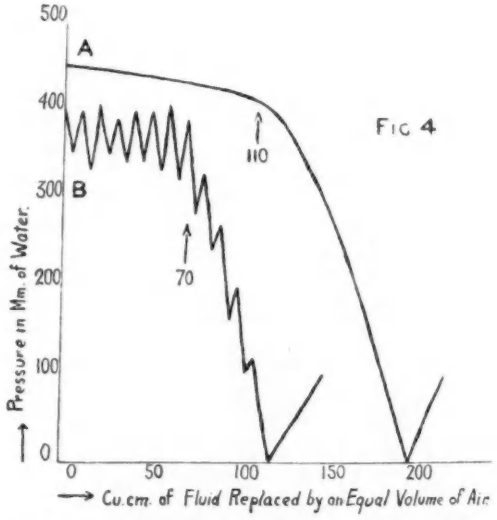

FIG. 4.—*Lumbar Cerebrospinal Fluid Pressures in Man during Lumbar Encephalography in the Sitting Position.* These curves demonstrate the marked change in *rate* of pressure decrease seen in many cases. The arrows indicate the point at which the cranium is emptied of cerebrospinal fluid. Curve A is that obtained by the simultaneous replacement method in a case with dilated ventricles and cerebral atrophy. Note the lack of pressure alternation when this method is used. Curve B is that obtained by the alternate replacement method with consequent alternation of pressures. In this case, alternations were relatively small. The intracranial volume was normal. Pressures in both cases were returned to 100 mm. by introduction of a final excess of air.

the inflammatory reaction was less marked, extravasation of blood into the fluid rarely occurred, the reaction of the patient was less severe, and the roentgenograms were more satisfactory.

(6) *Relation of the temperature of the gas to the pressure produced.*—The temperatures of all the patients at the onset of encephalography were normal (37° C.) and the room air introduced averaged 20° C. As was noted above, regardless of the value of A/F, even when an excess of air was introduced in respect to the volume of fluid removed, the lumbar arachnoid pressure decreased in all cases, approaching atmospheric

pressure regardless of the method of introduction. In no case was the increase in pressure observed, which might be expected, due to the attempt of the gas to expand as it was raised from 20 to 37° C. In a group of cases in which the simultaneous replacement method was used the air was heated by passing it through a hot water-bath before its entry into the subarachnoid. This procedure had no significant effect upon the resultant pressures.

(7) *Effect of encephalography on the composition of the cerebrospinal fluid.*—Samples of cerebrospinal fluid were taken previous to, during, and after encephalography and subjected to chemical and cytological study. In general, a lymphocytic and neutrophilic pleocytosis occurs in the cerebrospinal fluid during and subsequent to encephalography. Red blood-cells may appear occasionally due to trauma. The protein is slightly lowered and the sugar content slightly elevated. The cellular reaction was less pronounced when the simultaneous replacement method was used. All changes usually disappeared within two days.

(8) *Pressures subsequent to encephalography.*—In twenty-two cases the lumbar cerebrospinal fluid pressure was measured with the patient recumbent from one-half to forty-eight hours after encephalography (Table I). In only three of these cases was the pressure elevated above its normal value. In one of these the patient was struggling and the observations are unreliable; in another the patient developed a subarachnoid hæmorrhage several hours after encephalography. In the third case, though the pressure was elevated (190 mm. of water), the patient suffered no untoward reactions. Four other patients had a somewhat stormy course after encephalography, but their pressures were rather lower than increased (150, 70, 30 and 30 mm. of water).

(9) *Reaction of the patient to encephalography.*—In eighty-two cases a general anæsthetic was used during encephalography. The anæsthetics used were avertin, 100 mgm. per kilo body weight per rectum, or nembutal, 3 to 10 gr. intravenously. In these patients, especially those anæsthetized with nembutal, the subsequent reactions were somewhat less severe than in the unanæsthetized patients. These anæsthetics have little or no effect on patients accustomed to the use of barbiturates. Therefore, in twenty-two cases light morphine and scopolamine narcosis was employed, and in fifteen cases no narcosis was used. These patients complained of headache during the introduction of the first 10 c.c. of air. Withdrawal of fluid occasioned no complaint.

The reactions encountered during the replacement of fluid were: Low pulse pressure, tachycardia, cyanosis, hypernœa, hyperhydrosis,

nausea and vomiting, and rarely a fall in blood-pressure. In cases in which the anæsthetic had occasioned a fall in blood-pressure, the introduction of the first few cubic centimetres of air caused it to return to normal. Subsequent to encephalography the same chain of phenomena were seen in lesser degree, added to which were headache, stiff neck, hyperpyrexia (102° to 104° F.) and low skin temperature. The signs and symptoms were much less apparent when the simultaneous replacement method was used. Low blood and pulse pressures, vomiting and chills were rare. The hyperpyrexia was diminished and shortened, and the headache was much less severe.

Shock occurred in eight cases during encephalography. All of these were subjected to the syringe method of alternate replacement; six patients exhibited high lumbar cerebrospinal fluid pressures, and four were "brain tumour suspects." Shock occurred after encephalography in four cases, three of whom were "brain tumour suspects," two exhibited high spinal-fluid pressures, and two were over 50 years of age. One of these cases was a 52-year-old "brain tumour suspect" who developed a subarachnoid hæmorrhage several hours after encephalography. The simultaneous replacement method was used in two of the brain tumour suspects; in these shock occurred. There has been no fatality in this clinic to date.

The temperature and the relative or total volumes of gas used had no bearing upon the patient's reactions during or subsequent to encephalography. Room air was used in all cases. Those cases that presented a severe reaction to the procedure were analysed carefully. In one case, the spinal fluid pressure was increased after encephalograpy. The clinical appearance of the cases resembled the combined picture of surgical shock with superimposed meningeal irritation. The cerebrospinal fluid cell counts were often high in such patients for more than forty-eight hours after encephalography. Cases with dilated ventricles tended to recover more slowly than those with normal-sized ventricles and with less comfort than "normals."

(10) *Roentgenographic results.*—The roentgenographic results were analysed in all the cases with especial reference to the degree of replacement of intracranial cerebrospinal fluid and the distribution of the air. It was found that the ventricular outlines as well as the cisternal and convolutional markings were demonstrated more completely and clearly in those cases in which the ratio A/F was definitely greater than unity. That is, the reliability of the roentgenograms varied directly with the relative volume of air introduced. Manipulation of the head was, of

course, an important factor in producing adequate replacement. In twenty cases, the air demonstrated incompletely the cerebral structures. None of these cases was done by the simultaneous method : in twelve the ratio A/F was less than 1 ; in four it was equal to 1, and in only three was it greater than 1. In four cases no air at all could be demonstrated in the head. In one of these the apparatus leaked, and in two air was demonstrated in the neck up to the cisterna magna, but no block could be demonstrated.

IV. Discussion.—Loman, Myerson and Goldman [16 to 19] have made an extensive study of the lumbar and cisternal cerebrospinal fluid pressures of man in the recumbent, erect and sitting positions as well as measurements of the internal jugular pressures. They feel that the lumbar cerebrospinal fluid pressure is a direct reflection of the venous pressure at that point. Their work is very suggestive but not conclusive.

Weed and his co-workers [20 to 29] have investigated the relationship of postural change to cerebrospinal fluid and venous pressure in animals. They have concluded that there are two factors determining the pressures. One is the meningeal element determined by dural elasticity, which is active in the vertebral canal but negligible in the cranium. The other is the vascular element which is modified by autonomic tone and elasticity of the vessel walls. Both of these factors act synchronously, but only the predominant factor is responsible for the measurable cerebrospinal fluid pressure according to the type and position of the preparation. They conclude that the vascular factor, which is a reflection of the intradural venous pressure, is responsible for the intracranial pressure in the head-down vertical position but that the meningeal factor, allowing dislocation of cerebrospinal fluid, is predominant in the normal head-up position. The use of occipital and sagittal punctures, and of quadripeds in their crucial experiments, detracts from the value of their observations in interpretation of the lumbar pressures in man.

The present observations have shown that although the lumbar cerebrospinal fluid pressure in the sitting position is always greater than in the recumbent position it bears no direct relationship to the latter. This suggests that the pressure in the erect position is not composed of the recumbent pressure plus some factor x, but is entirely due to changes subsequent to postural alteration. Because of the wide variability of the relationship of the two pressures it is unlikely that the lumbar cerebrospinal fluid pressure in the erect position is largely determined by dural elasticity. Clinical observation of the patients

during encephalography suggested a wide range in venous pressures during the procedure. It was also observed that although in the majority of cases the vertical lumbar-cisternal distance was greater than the lumbar cerebrospinal fluid pressure, in some they were equal and in others the lumbar cerebrospinal fluid pressure was greater than this distance. This suggests that the column of cerebrospinal fluid from the needle to the cistern is not the determining factor. Here again the wide range suggests some facile factor. It is suggested, therefore, that the lumbar cerebrospinal fluid pressure of man in the erect position may be determined by the lumbar intradural venous pressure in the smaller veins and venules at that level.

Removal of cerebrospinal fluid will produce a progressive venous dilatation until restrained by the elasticity of the vessel walls. At this point the system becomes rigid toward sub-atmospheric pressures and cerebrospinal fluid will not run freely from the lumbar subarachnoid. The system is, therefore, not completely elastic. By introduction of air at this point, or by alternate replacement, the system may be emptied of cerebrospinal fluid. As air is replaced at atmospheric pressure the lumbar cerebrospinal fluid pressure must, therefore, progressively decrease until, at complete replacement, the pressure is atmospheric at the lumbar sac. This was observed to be true. At this point all veins in the system in which the pressure is above atmospheric, must be dilated. A certain proportion of the intracranial veins may not dilate, however, as their pressure is lower.

It was observed that alternate removal of cerebrospinal fluid and replacement with air, produced alternating decreases and increases in the lumbar cerebrospinal fluid pressure proportionate to the volumes replacing each other (fig. 2). Such pressure changes are transmitted in the cerebrospinal fluid system to the intracranial cavity. Thus each decrease would produce venule dilatation and cyanosis and possibly arteriolar constriction as has been demonstrated in cats by Forbes [30]. Increase in pressure reverses the procedure and hence a cerebral "massage" occurs during replacement. This is conducive to hyperæmia, œdema and hæmorrhage.

It was deemed advisable to produce a standard intracranial pressure during roentgenography in order that comparative observations may be made in various individuals from time to time. Without such a standard, encephalograms are not comparable even in the same individual. This standard was determined as the intracranial pressure normal for that individual at the time of encephalography when measured in the *recumbent* position.

When the cerebrospinal fluid has been "completely" replaced by air, the ventriculo-subarachnoid system is no longer a hydrodynamic but an aerodynamic or gas system in which pressures at all points are equal. Hence, the lumbar subarachnoid pressure is equal to the intracranial pressure. As the gravitational effect on such a system is infinitesimal the pressures remain the same regardless of the angle of the system with the horizontal. It will be seen, therefore, that if the final lumbar subarachnoid pressure, measured with the patient erect, be made equal to the lumbar cerebrospinal fluid pressure measured in the recumbent position previous to encephalography, the "normal recumbent" intracranial pressure will be produced with the patient erect.

In maintaining such a pressure, the ratio of air replaced to fluid removed (A/F) was quite variable, but in the great majority of cases A/F was greater than unity. It was observed that not only was there no increased intracranial pressure following replacement of equal volumes, but also there was no increased intracranial pressure when the replacing air exceeded the fluid removed (A/F greater than unity).

The replacement of any given volume of fluid from a rigid container at 37° C. with an equal volume of gas at 20° C. (room temperature) would produce an increased pressure. It has been apparent to all observers that the cranio-vertebral container of the cerebrospinal fluid system is semi-rigid in character. It was observed that assumption of the sitting posture by man results in an expression of an additional volume of cerebrospinal fluid into a lumbar manometer. Consequently, the measurable increase in lumbar spinal fluid pressure is merely confirmatory evidence of the non-rigid character of the container. A rigid container less than 32 ft. in length filled with fluid approximating the specific gravity of water (1·007) would not allow the fluid to run from it when changed from the horizontal to the vertical position. The observation that free flow of fluid from the subarachnoid ceases when atmospheric pressure is reached and *before* the subarachnoid is emptied confirms the views that the container is not completely elastic (open to atmosphere). A completely elastic container could not support such sub-atmospheric pressures as must exist at levels above the lumbar needle after flow has ceased. Therefore, being neither rigid nor elastic the system must be semi-rigid. The elasticity of such a system depends either upon the meningeal or vascular element [20 to 29]. Such elasticity is an individual variant.

Measurements of the cerebrospinal fluid pressure during and after

encephalography have conclusively demonstrated that it is *not* necessary to withdraw more fluid than the volume introduced in order to prevent an increased intracranial pressure. It was observed that the lumbar cerebrospinal fluid pressure decreased during encephalography regardless of the amount of gas introduced relative to the volume of fluid removed. There are two factors responsible for the prevention of increased intracranial pressure. The oxygen component (20 per cent.) of the air introduced is absorbed rapidly, thus reducing intracranial volume and pressure. Even if absorbed instantly this 20 per cent. decrease in volume would only neutralize the effect of the excess air introduced, as the volume of air introduced was often 20 per cent. greater than the volume of fluid removed. The second factor is that expansion of the gas, as it is raised to body temperature, is possible in the semi-rigid cranio-vertebral container of man. Expansion occurs by means of a diminution in intravascular volume within the system. Only 5 c.c. of blood need be expressed from the entire cranio-vertebral vascular system to allow for the replacement of equal volumes when introduced at atmospheric pressure and room temperature. Such a loss of blood volume does not appear to be hazardous. Both expansion and absorption are at work; which factor predominates is only of academic interest. The fact remains that throughout this series the volume of air introduced was often greater than the volume of fluid it replaced without the production of increased spinal fluid pressures or undue signs and symptoms.

As the elasticity of the cranio-vertebral container is an individual variant, volumetric changes cannot be used as indices of the pressures produced thereby. The cerebrospinal fluid pressures must be directly observed. We may therefore cease to pay much attention to the *volume* of air introduced in encephalography, focusing attention on the pressures themselves.

It must not be inferred that the production of a final lumbar subarachnoid pressure in the sitting position, which is equal to the normal pressure lying down, does not increase the usual intracranial pressure of the patient while sitting up. All pressures are equal in the gas-filled system and hence during roentgenography the intracranial pressure is at the normal level for recumbency (about 130 mm. of water) instead of at its normal of about minus 100 mm. of water. Thus, roentgenograms are made of the ventriculo-subarachnoid system under the pressure and volume relationships which exist normally in the recumbent position. A much more accurate picture is thus produced,

as vascular engorgement of the subarachnoid space is prevented. The roentgenograms are much better than by the older procedure and "incomplete filling" is a rarity.

At the conclusion of encephalography, when all the fluid has been replaced, the ventricular, cisternal and lumbar pressures are identical whether the patient be sitting or recumbent. Therefore, if a normal lumbar subarachnoid pressure be produced in such a system while the patient is sitting up, the pressure will remain the same when the patient is returned to bed. Consequently, the ventricular pressure will remain the same and the patient will be returned to bed with the same intracranial pressure which existed before encephalography. The measurements of the final lumbar pressures with the patient erect (156 mm.) and the final lumbar pressures with the patient lying down (140 mm.) show this to be true. When any difference occurred, the lumbar pressures in the recumbent position were slightly less (11 mm.) than the final pressures when sitting up. This is due either to incomplete drainage, in which case the small amount of fluid exerted a pressure in the sitting position, or to the fact that the venous pressure decreased in the recumbent position.

The results of studies on the cytology and chemistry of the cerebrospinal fluid during and subsequent to encephalography present additional evidence that simultaneous replacement produces less physiological disturbance than alternate replacement. When the alternation of pressures is removed by use of this method, red blood cells are rarely found in the spinal fluid, while leucocytes average 15 to 20 per c.c. rather than 200 to 400 by the former method. It would thus appear that much less meningeal irritation results from the simultaneous replacement method.

After encephalography has been completed, two series of reactions are set up which determine the subsequent intracranial pressure. The presence of the air with its consequent meningeal irritation very likely produces an increased rate of production of the cerebrospinal fluid. The removal of fluid also increases its rate of production. These factors are conducive to an increased intracranial pressure. On the other hand, the absorption of the air tends to reduce the pressure. Its oxygen content (20 per cent.) is absorbed rapidly, the inert gases more slowly. In addition, if the patient is in a state of shock (which is unusual) the venous pressure is low, thus increasing absorption. These factors tend to decrease the intracranial pressure. The pressure subsequent to encephalography is determined by the relative value of these

forces. The observations presented in this investigation demonstrate that the forces tending to decrease the subsequent cerebrospinal fluid pressures are predominant.

Even in the patients who presented a severe reaction subsequent to encephalography, measurement of the cerebrospinal fluid pressure demonstrated that the pressure usually was decreased rather than increased.

Analysis of the clinical features of the patients' reactions to encephalography show them to fit much better into the picture of surgical shock combined with a meningeal reaction. The low blood and pulse pressure, tachycardia and cold sweat suggest shock, while the hyperpyrexia, stiff neck, headache and vomiting are indicative of meningeal irritation. The cerebrospinal fluid pressures were not increased.

During the measurements of the lumbar cerebrospinal fluid pressure while the cerebrospinal fluid was being replaced volume for volume by syringe it was noted that the pressure decreased slowly at first and then suddenly began a rapid descent. This would be expected if the height of the column of fluid were a factor in producing the pressures. For every volume of fluid replaced from the relatively greater intracranial fluid spaces, the decrease in the height of the column would be less than when an equal volume of fluid was being replaced from the slender vertebral tube. It may be inferred that the change in rate of decrease of pressure occurs when the intracranial cerebrospinal fluid spaces have been emptied. It is theoretically possible, therefore, to accurately estimate the volume of intracranial cerebrospinal fluid in man. The procedure is as follows :—

Perform a lumbar puncture with the patient sitting erect and replace the cerebrospinal fluid by air, volume for volume, measuring the lumbar cerebrospinal fluid pressure after the introduction of each volume of air. Note the point where the pressure begins to decrease rapidly and continue replacement to confirm this observation. The volume of cerebrospinal fluid removed at this point should then equal the intracranial cerebrospinal fluid volume. The head should be manipulated during replacement to assist in complete evacuation of the ventricles.

During encephalography it should, therefore, be possible to obtain complete replacement of intracranial cerebrospinal fluid without continuing the replacement of intravertebral fluid. In a number of cases, this procedure was followed by excellent roentgenographic results.

Replacement was stopped when the pressure began to decrease rapidly and consistently. Either syringe or simultaneous methods are applicable. Several unsuspected cases of ventricular dilatation and cerebral atrophy were thus diagnosed previous to roentgenography by observing the volume of intracranial fluid. These suspicions were confirmed by roentgenography.

V. APPLICATION.—Many of these principles have been applied to the practice of encephalography with excellent results. These have been discussed in a separate investigation [32] and are briefly outlined herein that their relation to the dynamics may be more apparent.

(1) A complete lumbar puncture study should be performed previous to encephalography and the lumbar cerebrospinal fluid pressure in the recumbent position recorded for use during encephalography. The studies of the dynamics, cytology, chemistry and serology of the cerebrospinal fluid obtained from this puncture may contra-indicate or render unnecessary the subsequent encephalography.

(2) Encephalography is contra-indicated in the presence of a lumbar cerebrospinal fluid pressure in the recumbent position greater than 200 mm. of water or when signs of increased intracranial pressure are apparent. It is dangerous when weak-walled anomalies of the cerebral vascular bed are present.

(3) Anæsthesia or deep narcosis is an aid to patient and operator. Ether, ethyl chloride, nitrous oxide and other inhalant anæsthetics produce a dilatation of cerebral vessels increasing the pressure and preventing adequate filling, and are, therefore, contra-indicated.

(4) The best approach to the subarachnoid space is by lumbar puncture. The single puncture method is recommended only in the case of infants or when a double puncture needle[1] is not available. The use of such a double needle or of two lumbar punctures is recommended as simultaneous replacement may be carried out only through two aperatures.

(5) Some form of simultaneous replacement is recommended. The automatic apparatus is easiest to use and controls all the necessary factors. Only by simultaneous replacement may the alternations of intracranial pressure be prevented and with them the untoward reaction of the patient.

(6) Ordinary air at room temperature is recommended as the gas of

[1] Manufactured by Randall-Faichney, Inc., 123, Heath Street, Boston, Mass.

choice. It may be filtered through hot or cold sterile water, if desired. The amount of gas used is a variable, depending entirely upon the pressures produced during replacement. It is, therefore, unnecessary for practical purposes to record the volume of air introduced.

(7) All obtainable cerebrospinal fluid should be replaced by a volume of gas which produces a lumbar subarachnoid pressure in the *sitting* position approximating to the previously recorded lumbar pressure for that patient in the *recumbent* position. In order to do so it is usually necessary to introduce a volume of air greater than the volume of fluid it replaces. By such means the encephalograms for any series of cases are given a common basis and are hence subject to comparative studies, a condition which does not now exist.

(8) During replacement the patient's head should be slowly and continuously antero- and postero-flexed in order to evacuate the ventricles. Lateral flexion is contra-indicated as it predisposes to unequal distribution of the supra-cortical air.

(9) Some supportive apparatus is of great value in managing the anæsthetized patient [33].

(10) The entire procedure from puncture to picture should be performed with the patient sitting erect. In cases of suspected temporal or occipital lobe tumours, the appropriate roentgen exposures may be made in the recumbent position after the exposures have been made while the patient is sitting up.

Conclusions.—A. It has been demonstrated that :—

(1) The cranio-vertebral container of the cerebrospinal fluid system of man is semi-rigid.

(2) The cerebrospinal fluid system is subject to pressure alterations dependent upon its angle from the horizontal and upon pressure changes in its vascular components.

(3) The lumbar cerebrospinal fluid pressure measured with the patient sitting up closely approximates the vertical cisterno-lumbar distance for that patient, and bears no relation to the pressure measured in the recumbent position.

(4) Since the elasticity of the cranio-vertebral system is an individual variant, volumetric computations cannot be used as indices of expected pressure changes occurring in the ventriculo-subarachnoid spaces. Observations of such pressures must be made directly.

(5) Replacement of cerebrospinal fluid (by syringe) through a

single lumbar puncture produces alternate elevation and depression of the cerebrospinal fluid pressure. Simultaneous replacement of cerebrospinal fluid prevents these alternations and minimizes the patient's unfavourable reactions.

(6) When the cerebrospinal fluid has been completely replaced by gas, the ventriculo-subarachnoid space is a gas-filled system in which pressures at all points are equal irrespective of position. Therefore, the lumbar subarachnoid pressure in such a system remains the same whether the patient be sitting or recumbent, and consequently the intracranial pressures are unchanged.

(7) The production of a final lumbar subarachnoid pressure, after complete replacement of the cerebrospinal fluid by a gas, which approximates the normal lumbar cerebrospinal fluid pressure for that patient when recumbent is not dangerous. It results in excellent roentgenograms under normal pressure-volume relationships and does not increase the patient's reaction.

(8) It is possible to introduce a volume of air at room temperature (20° C.) greater than the volume of cerebrospinal fluid removed (at 37° C.) without significantly increasing the intracranial pressure.

(9) The introduction of a volume of air greater than the volume of fluid it replaces increases the reliability of the roentgenograms.

(10) Subsequent to encephalography, after the manner outlined, the cerebrospinal fluid pressure is not increased above its normal value. The untoward reactions occasionally occurring are not due to increased intracranial pressure.

B. It is highly probable but as yet unproven that :—

(1) Alternate replacement of cerebrospinal fluid by a gas through a single aperture produces alternate vasoconstriction and vasodilatation of the cerebral vascular bed with hyperæmia, œdema, hæmorrhage and severe reactions on the part of the patient.

(2) The reactions of the patient subsequent to encephalography are not due to increased intracranial pressure, but to shock and meningeal irritation.

(3) When the cerebrospinal fluid begins to decrease rapidly during replacement of the fluid by an equal volume of gas, the volume of fluid removed is then equal to the volume of intracranial cerebrospinal fluid.

The proper performance of encephalography depends not so much on the technique or apparatus as upon a thorough understanding of the underlying dynamic principles and their physiological results.

TABLE I.

LUMBAR CEREBROSPINAL FLUID PRESSURES DURING AND AFTER ENCEPHALOGRAPHY.

LUMBAR CEREBROSPINAL FLUID PRESSURES:

No of c.cm.		Csf. minus Air	Initial		Final		After Encephalography							Reaction of Patient
Csf.	Air		Rec.	Sit.	Sit.	Rec.	½ hr.	1 hr.	4 hrs.	6 hrs.	12 hrs.	24 hrs.	48 hrs.	
130	105	— 25	110	450	150	150		30						Poor.
120	100	— 20	110	350	100							30		,,
120	100	— 20	130	400							70			,,
115	95	— 20	130	420	250	200				80				Good.
135	120	— 15	170	140								80		,,
110	95	— 15	200	400	200	200					140			,,
87	75	— 12	100	375	110	100	30	20						,,
30	20	— 10	110	300	120	110				110			50	,,
110	100	— 10	150	475						0				,,
120	115	— 5	150	400	150	140				90				,, [1]
70	65	— 5	85	400	100	90					190			,,
100	125	— 5	110	400	130	130			110					,,
100	100	0	90	375	200	180							120	,,
50	50	0	150	400	150	150				150			50	Poor.
70	75	+ 5	100	500	200	150				110		50		Good.
100	110	+ 10	110	450	150	140	20							,,
107	117	+ 10	190	420	200	190						190		,,
90	100	+ 10	175	450	200	175			175	190		210		Poor.[2]
100	120	+ 20	130	550	130	120					310	400		Shock.[3]
95	115	+ 20	150	350	250	200		150		200		85		Good.
120	145	+ 25	130	440	150	150			30					,,
130	160	+ 30	150	400	150				70					,,

Csf.—Number of c.cm. of Cerebrospinal Fluid removed.

Air.—Number of c.cm. of Air replacing Fluid.

Rec.—Pressure with Patient Recumbent.

Sit.—Pressure with Patient Sitting.

Initial.—Pressure measured immediately previous to Encephalography.

Final.—Pressure measured immediately after Encephalography.

All other pressures were measured from ½ to 48 hours after encephalography and all were measured with the patient recumbent.

[1] Increased pressure but good recovery.

[2] Patient struggling during Pressure Measurement.

[3] Subarachnoid Hæmorrhage after Encephalogram.

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Notices of Recent Publications.

Indications Opératoires D'urgence dans les Lésions Traumatiques Fermées Récentes du crane et de L'encéphale. Par A. Jentzer. Paris: Masson et Cie. 1935. Pp. 105. Price 25 francs.

In this monograph, Jentzer has analysed all the cases of head injury which have entered the surgical clinic of the University of Geneva between the years 1914 and 1934. The bulk of the monograph is occupied by the brief descriptions and statistical analyses of these cases, which numbered 837.

The author summarizes his experience. More than 50 per cent. of the cases, many of them grave, recovered without surgical intervention. He regards this as a triumph for the conservative method.

He also sets out the indications for surgical intervention. These are the appearance of the following signs after a "free interval" of varying duration during which simple coma without localizing signs has been present: *Unilateral mydriasis.* This is invariably associated with a homolateral hæmatoma or gross cerebral lesion. *Localizing signs,* such as cranial nerve palsies, hemiplegia, monoplegia, Jacksonian fits, and a unilateral Babinski response. *Unilateral exophthalmos.* The progressive development of areflexia, slow pulse, loss of pupillary light reaction, fever, intracranial hypertension, persisting coma and stertorous breathing.

This careful record of the extensive practice of a single clinic by one observer lends the monograph a value and importance which must commend it to the neurosurgeon.

Les Syndromes Neuro-hématiques. Par H. Roger et J. Olmer. Paris: Masson et Cie. 1936. Pp. 225. Price 32 francs.

Professor Roger and his collaborator have in this monograph given a concise summary, clinical and pathological, of the various affections of the nervous system that are found in association with altered blood states.

Appropriately, the largest section is devoted to subacute combined degeneration of the cord, its ætiology, symptomatology, course, morbid anatomy and treatment.

Other sections deal with polycythæmia, the leukæmias, lymphadenoma, and the effects upon nervous function of severe hæmorrhage.

The reader will find the monograph comprehensive and embodying the most recent advances in knowledge.

Les Encéphalites Aiguës Post-Infectieuses de L'enfance. Par THÉRÈSE COMBY. Paris: Masson et Cie. 1935. Pp. 172. Price 30 francs.

The author's description is based upon a large series of personally observed cases of post-infective encephalitis and upon a review of the relevant literature.

The monograph is in the nature of a critical review of the subject, and will be found clear and comprehensive.

Les Hydrocéphalies Aiguës et Subaiguës d'origine Otique. Par R. BOURGEOIS. Paris: Masson et Cie, 1935. Pp. 178. Price 32 francs.

Under this title the author gives a comprehensive account, together with clinical summaries of all recorded cases, of the condition described in this country by Symonds under the name of "otitic hydrocephalus" (see this Journal, 1931, 54, 55). Otitic hydrocephalus is a rare complication of otitis media, usually of chronic otitis and in a young person. It is characterized by an acute onset and the appearance of the following signs: Papillœdema, a normal cerebrospinal fluid under increased pressure, headache and sickness. The author describes three forms, a diffuse cortical form, an encysted ventricular form and a form localized in the posterior fossa.

The symptomatology and differential diagnosis of these forms are discussed at length. Treatment by lumbar, cistern and ventricular punctures is described.

The monograph is a very comprehensive one, and will be of value both to otologists and to neurologists.

Die Welt der Geisteskranken. von K. BIRNBAUM. Berlin: Springer. 1935. S. 157. Price M. 4.80.

Most popular accounts of psychiatry revolt any psychiatrist who may look into them, and antagonize or mislead other intelligent readers. Professor Birnbaum's little book is an outstanding exception. With a command of the subject and a clarity of presentation that have made his scientific writings notable, he gives here an illuminating account of the natural history of sanity, touching more on the inner happenings than on the superficial phenomena. It is fresh in its mode of developing the theme, and informative even for psychiatrists. It would indeed be an excellent introduction to modern psychiatry for physicians, as well as for the larger public to whom it is addressed.

Emotions and Bodily Changes. A Survey of Literature on Psychosomatic Relationships. 1910-1933. By H. F. DUNBAR. London: H. Milford, Oxford University Press. 1935. Pp. 595. Price 25s.

A reference book such as this is valuable in direct proportion to the conscientious labour expended on its compilation. Dr. Dunbar's labour has been prodigious. A classified bibliography, running to 2,251 titles and covering 125 large pages, is plain evidence of the pains she has taken in collecting the material, and 430 tightly packed pages of summarizing text enable the reader to save himself much effort. The notices do not aim at being critical or exhaustive, but it is possible from them to decide about consulting an original article, and to get an orientation in this confused field. The survey is in places incomplete but some selection was inevitable. The first part is devoted to problems of integration, psychophysiological mechanisms in disease, and measurement; under the last head the psychogalvanometer and biochemical methods are included, as well as some reference to the "Berger rhythm." The second, and major part of the text, deals with the different organ-systems in turn, and is followed by brief therapeutic considerations and some concluding remarks.

Pellagroide Dermatosen an Geisteskranken. By P. J. REITER and J. JAKOBSEN. Kopenhagen: Levin and Munksgaard. 1935. Pp. 125.

Among nearly a thousand patients with mental illness in the St. Hans Hospital, Denmark, no less than a fifth were found to show dermatoses sufficiently like that of pellagra to raise the question of their nutritional origin. The authors consequently examined as controls a comparable number of convicts, who had lived in confinement for as long as the patients, and had been on a diet which was much poorer in vitamins and less varied than that given in the hospital. Among the convicts the number with pellagroid dermatoses was much smaller, and whereas there were fourteen insane patients with definite pellagra, at most only two of the convicts showed the disease. The cause for this difference was therefore assumed to lie in the patients rather than in their environment and diet. It was found that a majority of those with pellagroid dermatoses had either encephalitis lethargica or dementia præcox. In the encephalitics and some of the schizophrenics the dermatosis was attributed to disease of the central nervous system, especially in the hypothalamic region. In other schizophrenics, disturbances in absorption from the alimentary tract were held responsible for the appearance of true pellagra: this was in conformity with some earlier work on intestinal function by Dr. Reiter. It is difficult to account for so much higher a proportion of these dermatoses and of pellagra itself than could be found in similar English population: it may rest on different diagnostic criteria. In the cases reported, the pigmented

hyperkeratotic plaques on the elbows and hands seem to have been given an excessive importance. The study is of interest in its bearing on the relationship between the skin and the mental state.

Mental Health; its Principles and Practice. By FRANK E. HOWARD and FREDERICK L. PATRY. New York and London: Harper Brothers. 1935. Pp. 551. Price 15s.

An elementary account of the psychobiology of Adolf Meyer is the groundwork of this rather verbose, but sound, manual of mental hygiene. The pedagogic side is given more prominence than in many books of the kind, and the mental health of children and adolescents is the chief theme, in accordance with the prophylactic aim evident in most of the chapters. The sensible, if somewhat conventional treatment, the simple mode of expression, and the guides to further reading and discussion at the end of each chapter, make this book a useful introduction to psychiatry in its broader constructive aspects.

Wach- und Wahrträumen bei Gesunden und Kranken. von Prof. F. KEHRER. Leipzig: Thieme. 1935. S. 72. Price M. 4.80.

The author properly remarks on the deficiencies of the literature bearing on the topic of day-dreams. It cannot, however, be said that his monograph supplies the lack. He gives a review of what others have written, from which some important American papers are omitted, and the only original material he offers consists of one case, with a pedigree, to which is appended the speculation about heredity now apparently de rigueur in German psychiatric writings. The study of day-dreaming in children and normal adults, which is so necessary for psychopathology, is inadequately covered, though there is a diffuse discussion of the fantasies of artists, poets and clairvoyants.

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EDITOR.